The Emotional Component of Chronic Headache and its Relationship to Perceived Pain Severity and Disability.

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This thesis has been composed by myself and the work contained herein is my own. No part of this work has previously been accepted for any other degree, nor is any part of it being concurrently submitted in candidature for another degree.

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Abstract

Chronic primary headache is a prevalent health problem worldwide, and its impact on mental health and psychosocial functioning is well documented. Studies examining the emotional component of chronic headache have indicated that headache sufferers present significantly higher levels of depression, anxiety and anger in comparison with headache-free individuals.

The main aim of this study was to examine the emotions associated with the experience of chronic headache and its relationship with headache severity and headache-related disability. In particular, it was proposed to identify the types of emotion that are prominent in this patient group, the role of anger in chronic headache, and what strategies headache sufferers use most frequently to regulate their emotions, in comparison with non-headache controls.

A total of 104 individuals took part in this study. Of those, 57 were chronic headache sufferers, and 47 constituted the control group. Participants were asked to complete a questionnaire package comprising a demographic cover sheet and a range of measures assessing headache-related disability, basic emotions, emotion regulation strategies, anger, depression and anxiety.

Results indicated that headache sufferers experienced higher levels of emotional disturbance (including depression, anxiety and anger) than headache-free individuals. Affective distress was found to be associated with perceived emotional and functional headache-related disability and, to a lesser extent, with headache severity. Depression symptoms, anger, and the use of internal-dysfunctional strategies were found to be significant predictors of headache-related disability, while the use of internal-dysfunctional emotion regulation strategies was found to be a significant predictor of headache severity. These findings suggest that negative emotions are an integral part of the experience of chronic headache and need to be addressed in treatment.

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1. Introduction

1.1. Overview

Pain is an unpleasant experience with which we are all familiar. The International Association for the Study of Pain (1986) has defined pain as a multidimensional concept encompassing sensory, cognitive-evaluative and affective-motivational dimensions. This definition acknowledges the fact that even though pain is primarily perceived as a physical sensation, due to its unpleasant nature it is also perceived as an emotional experience. Emotion is perceived as fundamental to the experience of pain and not a reaction following the sensation of pain. Indeed, research into the emotional component of pain suggests that it is predictive of pain severity, pain-related disability and amount of medication patients use (e.g. Wade, Price, Hamer, Schwartz & Hart, 1990).

Studies examining the affective component of pain have identified anger, fear and sadness as a triad of negative emotions associated with chronic pain (e.g. Arena, Blanchard & Andrasik, 1984; Fernandez & Milburn, 1994; Greenwood, Thurston, Rumble, Waters & Keefe, 2003). Most studies, however, have focussed almost entirely on depression and (to a lesser extent) anxiety, while the study of anger, as an affective component of chronic pain, has been far less investigated. Nonetheless, a small body of research suggests that anger is one of the most prominent emotions associated with pain (cf. Fernandez & Turk, 1995; Greenwood, Thurston, Rumble, Waters & Keefe, 2003). Accordingly, one of the aims of this study is to explore further the emotion of anger in primary chronic headache, a subtype of chronic pain.

Chronic headache is a prevalent health problem, affecting individuals across a wide age span (cf. Boardman, Thomas, Croft & Millson, 2003; Holroyd, Malinoski, Davis & Lipchik, 1999; Sillanpaa & Anttila, 1996; Waldie & Poulton, 2002). The negative impact of chronic headache on a person's life has been well documented in relation to health and psychosocial functioning. For instance, chronic headache sufferers have been found to have poor health-related quality of life (Gardella & Pendino, 1997; Passchier, de Boo, Quaak & Brienen, 1996), marital relationship difficulties (Schwartz, Slater, Birchler & Atkinson, 1991) and decreased work effectiveness (Schwartz, Stewart & Lipton, 1997). Furthermore, the impact of chronic headache is also compounded by headache patients' frequent utilisation of medical care, which has cost implications for the health service (Boardman, 2003; Waldie & Poulton, 2000).

Although some research has examined the coping mechanisms of chronic headache sufferers (e.g. ter Kuile, Spinhoven, Linssen & van Houwelingen, 1995; Materazzo, Cathcart & Pritchard, 2000), there is a lack of research addressing the specific issue of how chronic headache sufferers regulate their emotions in general. Identifying the basic emotions associated with pain, as well as the emotion regulation strategies used by chronic headache sufferers, may help to tailor intervention that promotes the development of effective coping mechanisms, thus reducing the impact of pain in these patients' lives.

The purpose of this introduction is to review the main aspects of the current knowledge on the relationship between emotions and chronic primary headaches. It begins with a description of the most influential models of pain mechanisms, in order

to contextualise the role of the emotional components of pain. However, before addressing the issue of how emotions and headaches might be related, it is important to understand what emotions are, how they are generated, and which strategies individuals use to regulate them. Therefore, the following two sections will focus on theories and research on emotion and emotion regulation. An outline of the relationship between pain and emotion will follow, focusing mostly on the association between anger and pain. Next, the headache phenomenon will be described and the link between chronic headaches and emotional states will be addressed.

1.2. Pain Mechanisms

Pain usually indicates that something is not right. For instance, it might be generated by tissue damage or simply by noxious stimuli such as extreme cold or heat, which results in nerve excitation. The affected nerves stimulate the neuromatrix in the brain resulting in the perception of pain (Melzack, 1999; Merskey & Bogduk, 1994). However, this simple mechanism is not sufficient to define or explain all pain experiences as sometimes pain perception does not result from tissue damage, indicating that tissue damage may represent only a part of the pain experience. Furthermore, individuals seem to react differently to similar types of tissue damage not only in the way they express their pain experience but also in respect to resulting behaviours and suffering (Melzack, 1999).

Pain can persist long after healing of the initial injury is complete, as in chronic pain and phantom limb pain (Melzack, 1999). This nonprotective and persistent type of pain rather than being a symptom, become part of the disease itself and may provoke emotional disturbances such as anxiety and depression (e.g. Polatin, Kinney, Gatchel, Lillo & Mayer, 1993; Romano & Turner, 1985). Indeed, research has demonstrated that the emotional (e.g. anxiety, depression) and psychosocial (e.g. relationship difficulties, job loss, social isolation) effects of pain are not only as important as the pain sensations themselves but may be even more devastating (e.g. Cassidy, Tomkins, Hardiman & O'Keane, 2003; Flor, Turk & Scholz, 1987; Fordyce, 1995; Magnusson & Becker, 2003; Waldie & Poulton, 2002).

1.2.1. The Gate Control Theory

Various models have been proposed to describe the experience of chronic pain and one of the most influential theories to date is the Gate Control Theory proposed by Melzack & Wall (1968). This theory integrates the physiological and psychological aspects of the experience of chronic pain. It suggests that 'nerve gates' in the spinal cord either allow or prevent pain messages' access to the brain. This mechanism is influenced by several factors including the intensity of the pain messages, competition from other incoming nerve signals – which may affect the priority of the pain message – and the production of endorphins, which may inhibit pain messages. In addition, the model proposes that, depending on an individual's emotional state, descending messages from cortical and sub-cortical structures may either amplify the pain signal or prevent its access by 'closing' the nerve gate. The physiology of this model has generated significant debate (e.g. Nathan, 1979; Price, 1987) but its influence in neurophysiological (e.g. North, 1989), pharmacological (e.g. Fordyce, Roberts & Sternbach, 1985) and psychological (e.g.Turk, Meichenbaum & Genest, 1983) treatments of chronic pain is well established.

1.2.2. The Neuromatrix Theory

More recently, Melzack (1999) proposed the neuromatrix theory, which is based on the assumption that the experience of pain is generated by distinctive patterns of nerve activation within a neural network, named 'body-self neuromatrix'. This theory proposes that each individual has a unique body-self neuromatrix which, being genetically determined, is the main determinant of whether the person experiences pain or not. This assumption accounts for the individual differences in pain experiences. However, this neuromatrix can be influenced by sensory experience and

learning. As in the Gate Control Theory, it is postulated that sensory and evaluative processes can exacerbate or suppress the pain experience. This also occurs as a result of activation or deactivation of endogenous opioids.

Within a diathesis-stress model framework, this theory proposes to explain the initial development of pain via an interaction between predisposing factors and acute stressors (Turk, 2002). Prior learning is seen as a factor that actively shapes the body-self neuromatrix by influencing interpretative processes as well as physiological and behavioural response patterns (Melzack, 1999). The development of chronic pain occurs as the pain becomes a stressor in itself which in turn exacerbates the pain experience. Additionally, it has been suggested that the experience of recurrent or ongoing pain may result in structural and functional changes which alter perceptual processing, thus contributing to pain chronicity even after the initial cause has resolved (Woolf & Mannion, 1999). The neuromatrix theory is relatively new and still requires systematic investigation.

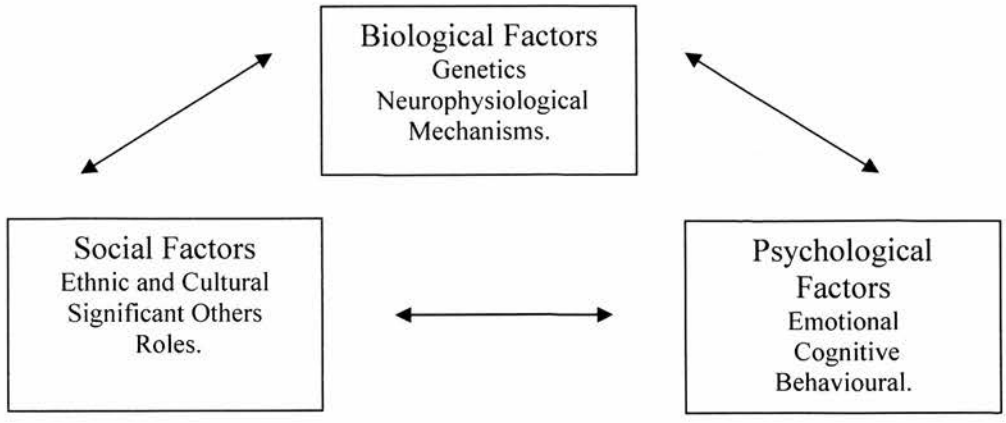
1.2.3. The Biopsychosocial Model of Chronic Pain

It is important to take into consideration that pain behaviours and suffering do not solely depend on the level of pain experienced. These elements are also influenced by personal and cultural factors, such as concurrent anxiety or depression, an individual's assessment of the significance of the pain and other people's reaction to their pain experience (Turk & Okifuji, 1999, 2002). A biopsychosocial model of chronic pain has been proposed as an attempt to explain pain and suffering through an understanding of the dynamic interaction of these factors (Turk & Okifuji, 1999, 2002).

This model proposes that the interaction of biological (e.g. genetics, neurophysiological mechanisms), psychological (e.g. anxiety, depression, beliefs, pain behaviours) and social variables (e.g. ethnic and cultural differences; friends and family reactions, roles) shapes an individual's perception and response to illness. It is assumed that the influence of these factors vary throughout the course of an illness or impairment. For instance, while in the acute phase biological factors are likely to prevail, over time psychological and social factors may play a more significant role in accounting for symptoms. Furthermore, it has been suggested that individuals differ considerably in the way these factors contribute to the manifestation of dysfunction, in fact, the relative contribution of these factors seem to vary within the same individual over time (Crook, Weir & Tunks, 1989).

In the biopsychosocial model of chronic pain, biological, psychological and social factors have a reciprocal relationship (Figure 1.1). While biological factors may initiate and maintain physical manifestations, psychological variables such as emotions and appraisals may influence a person's perception of internal physiological symptoms and trigger pain behaviours, which in turn may also be influenced by social factors.

Figure 1.1. Reciprocal relationships within the biopsychosocial model of chronic pain.



Research has demonstrated that psychological factors may affect biological processes such as hormone production (Bandura, O'Leary, Taylor, Gauthier & Gossard, 1987) and autonomic responses (Flor, Turk & Birbaumer, 1985). Pain behaviours may also affect biological processes. For instance, pain may be maintained by negative reinforcement as in when a person attempts to escape from pain by avoiding engaging in certain activities that are thought to increase the pain and cause further injury. Repeated avoidance behaviour may lead to physical deconditioning, which in the long term contributes to exacerbating the pain experience (Fordyce, Fowler, Lehmann & DeLateur, 1968; Fordyce, Fowler, Lehmann, DeLateur, Sand & Trieschmann, 1973; Sanders, 2002), and to anticipatory anxiety, which maintains the avoidance behaviour (Fordyce, Shelton & Dunmore, 1982; Phillips, 1987).

The role of social factors in the experience of pain has generated some debate in the literature. On one hand, there is evidence that social support is desirable and beneficial as it helps chronic pain sufferers to reduce levels of distress and inhibit avoidance behaviours, thus reducing disability (e.g. Cohen & Syme, 1985; Cohen &

Wills, 1985; Keefe, Smith, Buffington, Gibson, Studts & Caldwell, 2002, Kerns & Turk, 1984). On the other hand, it has been argued that sometimes support from significant others can have a detrimental impact on rehabilitation, as unhelpful pain behaviours may be positively reinforced by attention (Flor, Turk & Rudy, 1989; Fordyce, Fowler, Lehmann, DeLateur, Sand & Trieschmann, 1973).

The role of cognitive factors has been well documented in the literature. For instance, it has been shown that cognitions influence reports of pain, level of disability and treatment outcome (Flor & Turk, 1988; Jensen, Turner, Romano & Karoly, 1991; Turk & Rudy, 1992). A person's belief about pain may determine their therapeutic goals and their motivation to reach them. Williams & Thorn (1989) identified three kinds of beliefs and these are related to the temporal characteristics of pain (e.g. duration), the nature of pain (e.g. mysteriousness), and attributions (e.g. self-blame).

Another type of belief that can affect pain experience and treatment outcome concerns an individual's perceived sense of control. Locus of control has been typified as either internal, reflecting a belief in personal control, or external, reflecting a belief that powerful others or chance have control (Crisson & Keefe, 1988). An external locus of control fosters the belief that the person does not have responsibility over treatment outcome, hence the importance of helping chronic pain patients to adopt an internal locus of control over pain for successful treatment (Lipchik, Milles & Covington, 1993).

Overall, a patient's belief that they have the ability to work through pain, reduce disability and influence treatment outcome is a indication of perceived self-efficacy. Research has demonstrated that high self-efficacy is a strong cognitive factor that influences adaptive psychological functioning of chronic pain sufferers (Dolce, Crocker & Doleys, 1986; Spinhoven, ter Kuile, Linssen & Gazendam, 1989).

The role of cognitions as an essential factor in the experience of chronic pain is best conceptualised in the cognitive behavioural model of pain. The cognitive behavioural theory of pain proposed by Turk, Meichenbaum & Genest (1983) suggested that the experience of pain elicits catastrophic interpretations about the significance of the pain. This triggers a fear of engaging in activities that could cause further injury. As a consequence, avoidance behaviours may ensue and contribute to an increase in pain, through deconditioning, and a decrease in self-efficacy beliefs. This in turn may lead to learned helplessness and depression. At this point a vicious circle may be created whereby cognitive errors or catastrophic beliefs maintain the pattern.

More recently, Sharp (2001) reformulated the cognitive behavioural model placing more emphasis on the role of cognitive attributions and beliefs in the maintenance of chronic pain rather than on behavioural factors such as avoidance. He suggested that patients' appraisal of their pain determine the level of disability encountered. Pain behaviours such as avoidance prevent disconfirmation of negative appraisals. Concomitantly, emotional factors such as anxiety and depression reinforce catastrophic beliefs (e.g. beliefs regarding an underlying pathology) and further generate cognitive errors, thus maintaining avoidance and perpetuating the cycle.

Sharp's (2001) reformulated model proposes that stress and iatrogenic factors (such as misuses of medication and medical investigations) contribute to exacerbating anxiety (Pither & Nicholas, 1991; Kouyanou, Pither,Rabe-Hesketh & Wessley, 1998). The model also acknowledges the role of meta-cognitions, suggesting that an individual's attempt to suppress or neutralise pain related thoughts may actually heighten the experience of pain (Sullivan, Rouse, Bishop & Johnston, 1997; Harvey & Bryant, 1998).

1.2.4. Summary

The usual association between pain and tissue damage is not sufficient to explain the complexity of chronic pain. Accordingly, various multidimensional models have been proposed to account for the experience of chronic pain. One of the most influential models is the Gate Control Theory, which emphasises the role of emotional states as one of the factors that can allow or prevent pain signals' access to the brain. The Biopsychosocial model of chronic pain, however, provides a more comprehensive understanding of chronic pain as it takes into account the interaction between biological, psychological and social factors in the experience of pain.

1.3. Emotion

1.3.1. On Defining Emotion

It has been over a century since William James (1884) asked the question "What is an emotion?" and to date no satisfactory answer has been provided. Power and Dalgleish (1997) presented a comprehensive review of over 2000 years of philosophical and psychological theories of emotions. This review outlined that the theories of emotion stem from two major traditions: Plato's influential dualistic theory of mind – with an earthly body and an ethereal soul – where feelings occur in a spiritual domain but are a result of bodily processes, and Aristotle's views that something cannot be properly understood unless its constitution and function are known. A key point in Plato's approach is that he viewed emotions as irrational forces in continuous conflict with reason. Aristotle, on the other hand, proposed that there is a functional element in emotions and that different beliefs lead to different emotions.

According to Power & Dalgleish (1997), the Platonic views influenced early psychological theories which held that emotional reactions should be understood essentially in physiological or somatic terms (e.g. James, 1890; Watson, 1913), whereas the Aristotelian views influenced the cognitive theories, such as Schachter and Singer's (1962) arousal-based theory, which proposed that emotion involved a cognitive interpretation of a given physiological state of arousal. Following the decline of Behaviourism, cognitive theories of emotion have become prominent in the psychology literature and are considered to provide better models of emotion and emotion generation (Power and Dalgleish, 1997).

1.3.2. Cognition and Appraisal in Emotion

The role of cognition in emotion has generated strong debates in the emotion literature. For instance, in relation to how emotions are generated, Zajonc (1980, 1981, 1984) advocated the primacy of affect while Lazarus (1982, 1984, 1991) advocated the primacy of cognition. Zajonc proposed that cognitive processes occur after a stimulus has been assessed affectively as either positive or negative. Lazarus stated that cognitive activity is a necessary pre-condition of emotion because a person needs to understand in some form that a given transaction has some sort of implication for their well-being. For Lazarus, cognition is both a necessary and sufficient condition of emotion. Furthermore, an emotion can influence subsequent thoughts and generate other emotions (Lazarus, 1991, 1999).

In general, contemporary theories of emotion suggest that the concept of emotion involves an interpretation and subsequent appraisal of an instigating event, which induces physiological changes leading to potential behaviour, and usually there is conscious awareness of some or all of these components (Power & Dalgleish, 1997).

A key element of the cognitive theories is that particular emotions can be distinguished as a function of the appraisals associated with them. For instance, it is generally accepted that anger is associated with an appraisal of frustration, sadness with an appraisal of loss and fear with an appraisal of threat (e.g. Power & Dalgleish, 1997). This, ultimately, is related to how events affect an individual's goals (Oatley & Johnson-Laird, 1987; Frijda, 1988; Power & Dalgleish, 1997; Lazarus, 1999).

Lazarus' early theory (1966) of stress and emotion proposed that two interacting mechanisms were involved in the appraisal process: primary appraisal and secondary appraisal. Primary appraisal refers to the individual's evaluation of a particular situation and its potential to affect his well being, whereas secondary appraisal is the process whereby the individual evaluates which coping resources and strategies are available. Lazarus' early theory of appraisal influenced many other models that attempted to explain the process of emotion generation (e.g. Smith & Ellsworth, 1985; Leventhal & Sherer, 1987; Oatley & Johnson-Laird, 1987; Power & Dalgleish, 1997). Years later, Lazarus (1991) revised his theory of cognitive appraisal to include the notion of 'core-relational themes', which implies that an individual's appraisal of a given transaction is unique to each specific emotion. He suggested that individuals may have innate mechanisms, termed 'action tendencies', which are linked with the appraisal of personal harm or benefit. These action tendencies are said to be the basis for physiological patterns related to specific emotions.

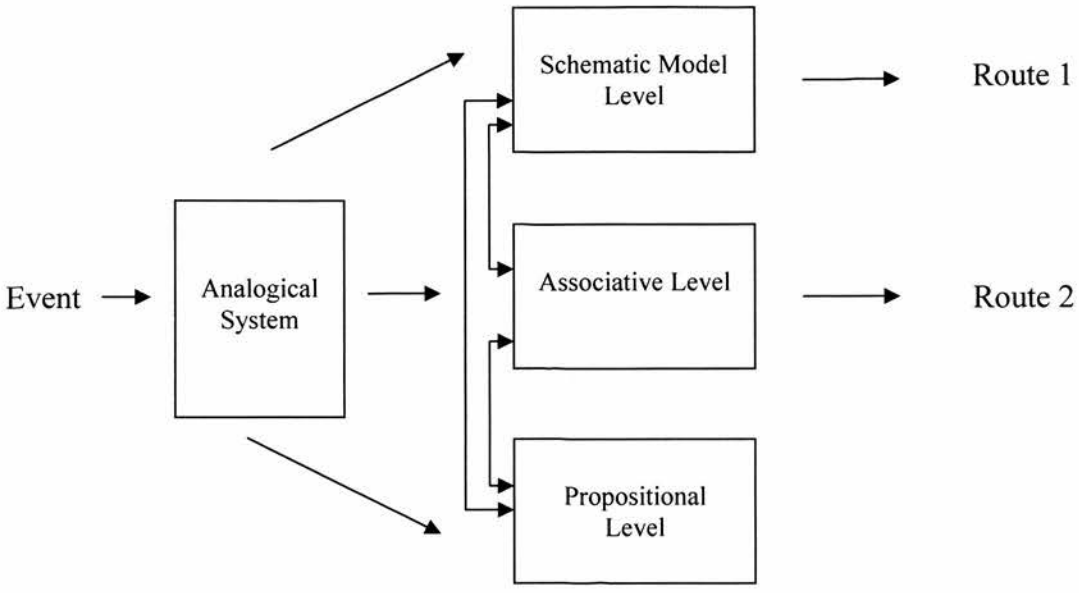
According to Scherer (1999), although the appraisal theories do not claim to present comprehensive models of emotion, they are the most dominant in the emotion literature. However, some appraisal theorists have been trying to address problems with the excessive emphasis appraisal theories place on cognition and have proposed that appraisal can occur at distinct levels of information and representation in the central nervous system (e.g. Leventhal & Scherer, 1987; Oatley & Johnson-Laird, 1987). In fact, there has been an increasing interest in the so called multi-level theories of cognition-emotion relations (cf. Teasdale, 1999), which will be briefly discussed in the next section.

1.3.3. Multi-level Theories of Emotion

In general, the multilevel-theories propose to view appraisal as a multi-level process. The main idea is that in order to understand the process of generation of emotions it is important to take into account, separately, the contribution from different types of information, and their interactions, that may occur at different levels of abstraction (Teasdale, 1999). Several multi-level theories have been proposed (e.g. Leventhal & Scherer, 1987; Barnard & Teasdale, 1991; Johnson & Multhaup, 1992; Power & Dalgleish, 1997).

The SPAARS (Schematic, Propositional, Analogical and Associative Representation Systems) approach is a relatively recent multi-level theory developed by Power & Dalgleish (1997, 1999). As implied by the name of the framework, the model is comprised of four representational systems: schematic model, propositional, analogical and associative, as shown in the diagram below.

Figure 1.2. Two routes to emotion within the SPAARS multi-level representation system (from Power & Dalgleish, 1997).



According to Power and Dalgleish (1997, 1999), the analogical representation system is where the initial processing of stimuli occurs, through mode-specific or sensory specific systems (e.g. visual, auditory, tactile, gustatory, proprioceptive and olfactory). The output from this processing feeds into the other three levels which operate in parallel.

The propositional representations are not regarded as a direct route to emotion – in contrast to the proposal in cognitive therapy in relation to automatic negative thoughts – instead, they feed either into the schematic level, via effortful appraisal, or directly into the associative level. In other words, emotions can be generated via two routes. Firstly, through an appraisal of goals at the schematic level of representation, which is seen as the highest level of semantic representation. Secondly, through the frequent experience of certain events, which in turn generate particular emotions and causes

the process of emotion generation to become automated through the associative level, whether or not they are processed at the schematic level. Furthermore, the model proposes that it is possible for the experience of a given emotion to be often paired or 'coupled' with the experience of another, which implies for example, that the first emotion, generated via the schematic level, could generate the other coupled emotion via the associative level.

Teasdale (1999) pointed out that the strength of the SPAARS approach is that it goes further than proposing an understanding of the generation of normal emotions. It also proposes an explanation of how disorders of emotion are generated. For instance, in relation to depression, Power (1999) suggests that depressed individuals may become locked or coupled in a cycle of sadness and self-disgust, where one emotion is continuously activating the other. This brings us to an important topic in the literature of emotions that is particularly relevant to this study: the notion of basic emotions.

1.3.4. Basic Emotions

Various emotion theorists have proposed that there is a set of basic emotions from which other more complex emotions stem (e.g. Plutchik, 1980; Levenson, Ekman & Friesen, 1990; Ekman, 1992; Oatley & Johnson-Laird, 1987; Power & Dalgleish, 1997). According to Ekman (1999), basic emotions are those which differ from each other with respect to various distinctive characteristics such as antecedent events, appraisal, and physiological patterns. Therefore, an emotion can be classified as basic when it cannot be further reduced once these and other components have been examined. One criterion used to determine basic emotions is the examination of

universal signals, such as facial expression, and physiological activity (Ekman, 1992; Levenson, Ekman & Friesen, 1990). However, Power & Dalgleish (1997) argued that the best way to distinguish basic emotions is to define a core set of universal appraisal scenarios - rather than universal signals or physiological patterns - for each basic emotion.

Not surprisingly there is substantial disagreement amongst emotion theorists as to which emotions are prototypical or basic and different "lists" have emerged (cf. Ortony & Turner, 1990). Taking into account several of these lists, derived from different theories and empirical evidence (e.g. Oatley & Johnson-Laird, 1987; Lazarus, 1991a), Power & Dalgleish (1997) proposed a list of five basic emotions that can be distinguished not only according to basic appraisals scenarios, but also according to other criteria such as universal signals and physiological activity. The five basic emotions they proposed are anger, fear, disgust, sadness and happiness.

According to Power & Dalgleish (1997), the basic emotion of *Anger* occurs when there is an appraisal of blocking or frustration of a role or goal, through a perceived secondary agent. *Fear* results when there is an appraisal of physical or social threat to the self or to a valued role or goal. *Disgust* occurs as a result of an appraisal of a person, object, or idea as being repulsive to the self and to valued roles and goals. *Sadness* results from the appraisal of an actual (or potential) loss, or failure, of a valued goal or role. *Happiness*, on the other hand, occurs when the appraisal process indicates a successful move towards (or completion of) a valued role or goal.

These basic emotions are said to be primarily adaptive, however they can become dysfunctional depending on contextual factors and on how frequently they are experienced (Power & Dalgleish, 1997). For instance, anger can result in aggressive behaviour (Berkowitz, 1990) and in anger related disorders that require treatment (Novaco, 1975, 1979). The emotion of anger, as well as anger regulatory strategies used by chronic headache sufferers, will be addressed in more detail in subsequent sections.

Fear is a basic component of many disorders described in the DSM-IV (American Psychiatric Association, 1994) such as generalised anxiety disorders, phobias, panic disorder and post-traumatic stress disorder. Power & Dalgleish (1997) pointed out that anxiety results from an individual's inability to deal with something that threatens their goals.

Rozin, Haidt & McCauley (1999) suggest that sensitivity to disgust may be related to psychopathology in some form. For instance, it has been suggested that complex emotions such as guilt and shame are derived from disgust that is directed towards the self (Power & Dalgleish, 1997). Guilt, for instance, is said to be implicated in obsessive-compulsive disorder (Rachman, 1993) and feelings of shame are implicated in social phobia (Beck & Emery, 1985). As to sadness, Power (1999) pointed out that this basic emotion is often experienced in conjunction with other basic emotions such as fear, anger or disgust. For example, some forms of depression can be conceptualised as the coupling of sadness and self-disgust. Similarly, grief can be the result of the coupling of sadness and anger. Happiness, on the other hand, is primarily perceived as a positive emotional state. However, as Power & Dalgleish

(1997) pointed out, a feeling of joy at another's misfortunes could be conceived as a negative response. In addition, there are also disorders of happiness such as mania and hypomania.

1.3.5. Summary

Theories of emotion are complex and controversial. For centuries philosophers and more recently psychologists have been attempting to develop theoretical frameworks and generate empirical evidence of what constitutes an emotion, what are its components, what processes occur in the generation of emotions, and which emotions are basic or prototypical. Cognitive appraisal theories seem to offer stronger theoretical and empirical arguments in the pursuit of this endeavour, but it is now clear that these theories must be developed into multi-level processes by means of which emotion can be generated by more than one route. Still, appraisal theorists differ in their views and the search for a better understanding of what constitutes an emotion continues. Five basic emotions have been proposed: anger, fear, disgust, sadness and happiness. It has been postulated that other complex emotions derive from these prototypic emotions. There are many issues in the emotional literature that have not been addressed above. The ones discussed are those which are essential for an understanding of how emotions can be regulated, which will be the subject of the next section.

1.4. Emotion Regulation

As discussed in the previous section, emotional experiences involve multi-level processes eliciting changes at abstract, behavioural and physiological levels. As Gross and Muñoz (1995) pointed out, the notion that emotions are powerful, and at times overwhelming or uncontrollable, is reflected in common expressions such as 'getting carried away by anger' or even in legal terms such as 'crimes of passion' as opposed to those committed in 'cold blood'. At the same time, it is part of human experience to exert control over emotions. The effort to sooth a child, the effort to contain rage or even laughter, the effort to hide feelings to avoid hurting another's self-esteem, the effort to cheer oneself up or make someone who is feeling sad laugh, are all examples of attempts to regulate one's own emotions or someone else's experience of unpleasant emotions.

It has been suggested that there is an element of truth in both ideas – the belief that emotions are uncontrollable and the belief that they can be controlled – indicating that some measure of control is needed (Gross, 1998a, 1998b; Gross & Muñoz, 1995). Coming from that perspective, researchers have attempted to identify how individuals manage their emotions and what results come from particular regulatory strategies. Indeed, it has been shown that the regulation of emotions is an essential feature of mental health (Cicchetti, Ackerman & Izard, 1995; Gross & Muñoz, 1995; Southam-Gerow & Kendall, 2002), yet research on emotion regulation is a relatively new field that has been developing only in the past two decades, initially in the developmental literature (e.g. Campos, Barrett, Lamb, Goldsmith & Stenberg, 1983; Campos, Campos & Barrett, 1989; Eisenberg, Fabes, Murphy, Maszk, Smith & Karbon, 1995; Thompson, 1991), and more recently in the adult literature (e.g. Garnefski, Kraaij &

Spinhoven, 2001; Gross, 1998a, 1998b, 1999; Gross & Levenson, 1993; Gross & Muñoz, 1995; Izard, 1990). In the following sections it is proposed to review some of this research but first it is important to clarify what is meant by emotion regulation, beginning with a brief discussion of how the concept developed in the literature.

1.4.1. Theoretical Precursors of Emotion Regulation

Theoretically, the main precursors in the study of emotion regulation are the psychoanalytic tradition and the stress and coping tradition. From the psychoanalytic tradition, the main influence is the concept of ego defense as an unconscious regulatory process of anxiety and other negative affects (A. Freud, 1946; Paulhus, Fridhandler & Hayes, 1997). According to Gross (1998b), two types of anxiety regulation are emphasised by this tradition. One refers to reality-based anxiety, which emerges when situational demands overwhelm the ego. In this case, anxiety regulation involves avoidance of similar situations in the future. The other type of anxiety regulation concerns id- and superego-based anxiety, which emerges when strong impulses urge for expression. In this case, anxiety regulation involves restricting the expression of impulses which the ego judges will create further anxiety. These ego defenses operate outside of awareness (Erdelyi, 1993) and individuals present stable defensive styles which differ in terms of reality distortion and unnecessary nongratification of impulses, consequently resulting in individual differences regarding impairment (Fenichel, 1945; A. Freud, 1946; Haan, 1977; Vaillant, 1977). Gross (1998b) pointed out that, while emotion regulation researchers remain interested in unconscious processes, their focus has expanded to include conscious processes that increase or decrease the experience or expression of negative and positive emotions. In Gross' (1998b) view, emotion regulation should

be thought of as a continuum from conscious, effortful, and controlled regulation to unconscious, effortless, and automatic regulation.

From the stress and coping tradition, the main influence is the emphasis on adaptive, conscious coping processes which shifted the focus from person variables to situational variables (Parker & Endler, 1996). According to Gross (1998b), emotion regulation researchers have been heavily influenced by the stress and coping tradition. In fact, it has been suggested that "all coping efforts by an individual come under the broad definition of emotion regulation" (Garnefski, Kraaij & Spinhoven, 2001, p. 1312). Emotion regulation researchers, however, have shifted the focus from the broad concept of stress to examine more specific emotions (Gross, 1998b). Still, it is important to examine the concept of coping first to gain a better understanding of the concept of emotion regulation.

1.4.2. Coping

Lazarus & Folkman (1984) defined coping as a cognitive or behavioural attempt to alleviate stress. However, the way an individual copes with a particular situation depends on what that event means to the individual, the context within which the event occurs and on the individual's goals (Lazarus, 1993). Lazarus & Folkman (1984) make a distinction between *problem-focused* coping and *emotion-focused* coping. Problem-focused coping involves managing or altering the cause of stress, whereas emotion-focused coping focuses on regulating the individual's emotional response to the problem.

The emotion-focused style of coping usually occurs when the individual perceives the problem as unchangeable. The person's coping strategies involve cognitive processes aimed at decreasing emotional distress and include strategies such as avoidance, minimisation and distancing (Lazarus & Folkman, 1984). This type of coping can be used to re-appraise situations in a way that elicits hope and optimism by changing the meaning of the situation. One of the problems with this type of coping is that sometimes it can lead into self-deception or distortion of reality (Folkman, Dunkel-Schetter, DeLongis & Gruen, 1986; Lazarus & Folkman, 1984; Lazarus & Smith, 1988).

The problem-focused style of coping is likely to occur when the individual perceives the situation as changeable. According to Lazarus & Folkman (1984), the cognitive processes involved are similar to those used for problem-solving although the effort is not only directed at changing the environment, but also directed at the processes occurring within the individual. These strategies could be aimed at altering environmental pressures or could be focused on motivational or cognitive changes such as creating new standards of behaviour or acquiring new skills.

In theory, both coping processes can either facilitate or impede each other (Lazarus & Folkman, 1984). Furthermore, there seems to be no consensus regarding which style of coping is more effective. For instance, Aldwin & Revenson (1987) pointed out that some studies have found that problem-focused coping helps to decrease emotional distress, while emotion-focused tends to increase it. However, some studies have described the opposite effect (Aldwin & Revenson, 1987; Felton & Revenson, 1984).

The terms coping and emotion regulation have been used interchangeably (e.g. Saarni, Mumme & Campus, 1998) but according to Gross (1998b, 1999), although the concepts of coping and emotion regulation overlap, it is important to stress that there are some significant differences between the two constructs. For example, coping processes may involve taking actions that are not necessarily emotional in order to achieve non-emotional goals. For instance, a problem-solving strategy such as map-reading when lost in an unfamiliar place is a form of coping, which may not necessarily involve emotion regulation (Scheir, Weintraub & Carver, 1986). Coping is therefore a broader category, whereas emotion regulation refers particularly to actions taken to achieve emotional goals (Gross, 1999).

1.4.3 On Defining Emotion Regulation

The concept of emotion regulation has been developing as research in this area expands. Two meanings of emotion regulation have been described in the literature (e.g. Southam-Gerow & Kendall, 2002). One refers to the regulation of something (e.g. behaviour) *by* emotions and the other refers to the regulation *of* emotions. The first meaning is related to an individual's response to environmental events. This means that the experience of an emotion is a regulatory phenomenon in itself. The second meaning refers to how individuals attempt to regulate their emotions, i.e., one's effort to influence which emotions they experience and how they should be expressed. Most research on emotion regulation is concerned with the latter construct (cf. Gross, 1998b, 1999).

Thompson (1994) defined emotion regulation as "all the extrinsic and intrinsic processes responsible for monitoring, evaluating and modifying emotional reactions,

especially their intensive and temporal features, to accomplish one's goals" (pp.27-28). This definition encompasses a wide range of regulatory processes within physiological, social and behavioural spheres including conscious and unconscious cognitive processes. Broadly speaking, it refers to the regulation of emotions by oneself or by others, and the regulation of emotions themselves as well as of their underlying features (Thompson & Calkins, 1996).

Within this perspective, various possible ways by which emotions are regulated have been suggested (Thompson, 1994; Garnefski, Kraaij & Spinhoven, 2001). These include intrinsic ones such as neurophysiological response, attentional processes, attributions, and other more extrinsic ones such as access to coping resources, exposure to environment and behavioural responses. For instance, in a physiological way, emotions are self-regulated by a rapid pulse, increased breathing or shortness of breath, sweat and other accompanying features of emotional arousal. Seeking interpersonal or material support are examples of regulating emotions in the social way. Behaviours such as screaming, crying or withdrawing are examples of strategies to manage emotions. Unconscious processes such as selective attention, denial, self-blame or blaming others, memory distortions, catastrophising and ruminating are also strategies to manage emotions (Thompson, 1994).

Gross (1998b) defined emotion regulation as "the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions" (p.275). These processes are said to be automatic or controlled, conscious or unconscious, and their effect may be felt at one or more points of the process of emotion generation. In accordance with other theorists (e.g.

Thompson, 1994; Masters, 1991) Gross initially included attempts to regulate others' emotions in his definition of emotion regulation (Gross & Levenson, 1993; Gross & Muñoz, 1995), however, he later changed his views on the grounds that the motives, goals and processes involved in these two processes are potentially different.

An important aspect in the literature is the distinction between regulation and control. According to Cole, Michel & Teti (1994), regulation refers to the dynamic process of ordering and adjusting emotional behaviour. Control, on the other hand, refers to the restraint of these processes. The main implication of this distinction is that emotion regulation goes beyond attempts to stop or reduce emotions. Indeed, it implies that individuals are able to increase, maintain and decrease both negative and positive emotions (Gross, 1998b). Ultimately, this means that emotion dysregulation does not imply a lack of regulatory abilities, instead it refers to strategies that are operating in a dysfunctional manner (Cole, Michel & Teti, 1994). In fact, it has been suggested that it is preferable not to make a priori assumptions as to whether certain types of emotion regulation are good or bad (Gross, 1998b; Thompson & Calkins, 1996). The idea is to avoid the confusion created in the stress and coping literature that predefined *defenses* as maladaptive and *coping* as adaptive (Gross, 1998b).

1.4.4. Emotion Regulation in the Psychological and Physical Health Literature

Emotion regulation has been considered in the literature concerned with both psychological and physical health. However, there appear to be different views in these two broad categories as to what are the consequences of emotion regulation (Gross, 1998a). Earlier studies by Lazarus and colleagues (Lazarus & Opton, 1966; Lazarus & Alfert, 1964), for example, showed that certain cognitive strategies could

reduce stress response, suggesting that this type of regulation might benefit psychological health. Gross (1998a) pointed out that even though this assumption has been incorporated into emotion theories (e.g. Frijda, 1988; Lazarus, 1991; Plutchick, 1980), coping and stress theories (e.g. Lazarus & Folkman, 1984) and psychopathology (e.g. Barlow, 1988), these studies have been difficult to replicate.

In relation to physical health, earlier studies depicted the regulation of negative emotions as something that could be detrimental to an individual (cf. Gross, 1998a). For instance, the inhibition of sadness and crying was thought to be linked to disorders of the respiratory system (Alexander, 1950; Halliday, 1937), and the chronic inhibition of anger was linked with cardiovascular diseases (Alexander, 1939). Although some of these ideas have now been disregarded, the association of the inhibition of anger and hostility with cardiovascular diseases continue to be popular in research (Diamond, 1982; Steptoe, 1993). Accordingly, Fernandez and Turk (1995) outlined that suppressed anger is strongly associated with chronic pain. Indeed, Franz, Paul, Bautz, Choroba & Hildebrandt (1986) found that low back pain and headache patients had a tendency to deny feelings of anger and aggressiveness in comparison with pain-free control subjects. It has been suggested that chronic pain patients may inhibit the expression of anger due to the social undesirability of this emotion (Fernandez and Turk, 1995). It has also been suggested that the inhibition of emotions is likely to exacerbate minor illnesses (Pennebaker, 1990) and that the lack of emotion expression may accelerate the progression of cancer (Gross, 1989).

According to Gross (1998a), these studies suggest that exerting tight control over negative emotions may affect physical health in an adverse way. The mechanisms

underlying this are not known but it has been argued that the inhibition of emotions exacerbate physiological responses that may cause damage in the long term (e.g. Krantz & Manuck, 1984).

As Gross (1998a) pointed out, an examination of these two broad literatures give the false impression that emotion regulation may be beneficial for psychological health and detrimental for physical health. In order to address this issue, he proposed a model whereby emotional response tendencies may be modulated, and this modulation is what shapes up the manifest emotional response. His model suggests two major ways to regulate emotions: *antecedent-focused* and *response-focused.* Antecedent-focused emotion regulation occurs before a given emotion is generated and concerns things individuals do before the onset of an emotion that affect whether on not the emotion occurs . Response-focused emotion regulation occurs after the emotion has been generated and the emotion component processes are already in place (Gross, 1998a, 1998b; Gross & Muñoz, 1995).

In an attempt to integrate these two divergent views of emotion regulation, Gross (1998a) suggested that antecedent-focused emotion regulation might be the primary concern of the psychological health literature, whereas the physical health literature is more concerned with response-focused emotion regulation. In other words, the former is concerned with regulation before the emotion is triggered, while the latter is concerned with regulation of emotion that has already been generated.

Gross (1998a) speculated that probably certain forms of antecedent-focused emotion regulation, such as reappraisal, may be more beneficial than certain forms of

response-focused emotion regulation, such as suppression. However, it has been suggested that different forms of emotion regulation might have different consequences depending on the context, which means that no strategy can actually been seen as superior across all situations, hence the importance of understanding the advantages and disadvantages of diverse regulatory processes to help individuals match their style of emotion regulation with situational demands (Gross, 1999a, Gross & Muñoz, 1995). Furthermore, specific emotions such as anger, fear, sadness and disgust might present different challenges in terms of emotion regulation strategies (Gross, 1998a). Indeed, there is physiological evidence that different emotions may be regulated in different ways (LeDoux, 1994).

1.4.5. Styles of Emotion Regulation

According to Gross (1998b) the concept of individual differences is central for an understanding of emotion regulatory processes. In the coping literature there is evidence to suggest that individuals acquire coping styles that are relatively stable over time (Gomez, 1997; McCrae and Costa, 1986) such as the emotion- and problem-focused strategies already discussed, although many other factors have also been identified (Parker and Endler, 1992). The relationship between coping styles and patients' adjustment to chronic pain will be discussed subsequently.

Kokkonen, Pulkkinen & Kinnunen (2001) recently reported on a longitudinal study looking at low self-control of emotions as an antecedent of self-reported physical symptoms in 112 men and 112 women. This study investigated personality characteristics indicating low self-control of emotions (e.g. emotional lability) at ages 8 and 27 and their association with self-reported physical symptoms at age 36. The

main purpose was to examine the mediating effect of *repair* – an emotion regulation strategy whereby one actively attempts to turn a negative emotion into a more positive direction (Mayer & Stevens, 1994). At age 8, emotional lability was indicated by inattentiveness, shifting moods, aggression and anxiety. At age 27, emotional lability was linked to neuroticism (Eysenck & Eysenck, 1975). Overall, results revealed that low self-control of emotions, specifically characterised by inattentiveness in childhood in boys and by shifting moods in girls, was an antecedent of self-reported physical symptoms in adulthood such as gastrointestinal problems in men and pain and fatigue in women. Neuroticism at age 27 was related to self-reported physical symptoms at age 36 but only in men. This was thought to be related to gender differences in the relationship between personality traits and coping strategies reported in other studies (e.g. Amirkhan, Risinger & Swickert, 1995; Gomez, Holmberg, Bounds, Fullarton & Gomez, 1999; Rim, 1993).

In relation to psychological functioning, Gross and Muñoz (1995) pointed out that emotion regulatory processes are central to three basic, and overlapping, domains of mental health: the ability to work, the ability to relate to others and the ability to enjoy oneself. Indeed, Gross & Levenson (1997) argued that emotion dysregulation is implicated in more than half of the DSM-IV Axis I disorders and in all of the Axis II disorders (American Psychiatric Association, 1994).

In one way or another, clinical interventions usually aim to modify ineffective patterns of emotion regulation. Gross (1998b) argued that what is crucial in this therapeutic process is the identification of what constitutes appropriate strategies and this will vary according to individual differences because this will determine not only

what one's emotion regulatory goals are, but also what they would like them to be and how these goals could be achieved.

The types of dysfunctional emotion regulation identified in the literature usually involve either the inhibition or the excessive expression of emotion (cf. Garnefski, Kraaij & Spinhoven, 2001; Gross, 1999; Pulkkinen, 1995, 1996; Southam-Gerow & Kendall, 2002). In general the inhibition of emotion involves the use of internal resources (such as cognition, or the use of substances that activate physiological response), whereas expressive strategies involve using external resources (such as other people or objects) to regulate emotions.

Based on current literature, Phillips (2003) developed a conceptual model of emotion regulation which proposes that dysfunctional strategies involve a rejection of unwanted emotional experiences, whereas functional strategies involve accepting these experiences and making use of the information they provide. In this model, emotional regulatory strategies were conceptualised as dysfunctional-versus-functional which draw on internal-versus-external resources. To illustrate this conceptual model, Phillips (2003) presented examples of dysfunctional-functional internal and external strategies found in the literature. These are detailed in the table below.

Table 1.1. Emotion regulation strategies identified in the literature and their place in Phillips conceptual model (from Phillips, 2003).

Strategies	Internal	External
Dysfunctional (Rejection of Emotion)	Denial (e.g. Freud, 1936)	Bullying (e.g. Caspi et al., 1995)
	Repression (e.g. Freud, 1915, 1957; Weinberger, 1990)	Verbally abusing others (e.g. Ciarocchi et al., 2003)
	Rumination (e.g. Nolen-Hoeksema et al., 1994)	Physically abusing others (e.g. Ciarocchi et al., 2003)
	De-personalisation/de-realisation (e.g. Phillips et al., 2001)	Making others feel bad (e.g. Hare, 1970)
	Emotion 'switch' (e.g. Power & Dalgleish, 1997)	Lashing out at objects (e.g. Ciarocchi et al., 2003)
	Binge-eating (e.g. Lingswiler et al., 1989)	Avoidance of situations (e.g. Gross, 1998b)
	Restricting food (e.g. Bruch, 1979)	Helping others: negative state relief (e.g. Cialdini et al., 1973)
	Substance Use (e.g. Rather et al., 1992)	
	Resignation/hopelessness (e.g. Seligman, 1975)	
	Self-harm (e.g. Gratz, 2003)	
	Negative social comparison (e.g. Allan & Gilbert, 1995)	
Functional (Acceptance of Emotion)	Positive reappraisal (e.g. Carver et al., 1989)	Social support: sharing feelings (e.g. Parkinson et al., 1996)
	Modification of goals (e.g. Oatley, 1992)	Social support: advice seeking (e.g. Parkinson et al., 1996)
	Planning (e.g. Kopp, 1989)	Social support: physical contact from others. (e.g. Parkinson et al., 1996)
	Concentration (e.g. Csikszentmihalyi, 1975)	Engaging in peaceful/relaxing activities (e.g. Gross, 1999)
	Upward social comparison (e.g. Taylor & Lobel, 1989)	Exercise (e.g. Thayer et al., 1994)
	Putting into perspective (e.g. Garnefski et al., 2001)	Change of environment (e.g. Gross, 1998b)
	Positive suppression (e.g. Myers et al., 2003)	

Based on this model, Phillips (2003) developed a measure of individual differences in emotion regulation as well as a measure of emotion regulation strategies used with specific emotions. In a recent study looking at emotion regulation, psychological health and quality of life in children and adolescents, Phillips (2003) found that internal-dysfunctional emotion regulation strategies were strongly associated with external-dysfunctional ones. This suggests that the inhibition of emotion relates to the under-control of emotional expression (Southam-Gerow & Kendall, 2002). By the same token, internal-functional emotion regulation strategies were strongly associated with external-functional ones, suggesting that individuals who accept and deal with emotions internally are more able to share these feelings externally. Interestingly, the frequent use of internal-dysfunctional strategies was associated with infrequent use of external-functional strategies. According to Phillips (2003), this indicates that the more an individual uses internal resources to reject or inhibit emotions, the less they use helpful external resources, such as other people, to express their emotions.

Regarding the relationship between emotion regulation and health, in a sample of 225 adolescents aged between twelve and nineteen, Phillips (2003) found a significant correlation between the self-reported use of internal-dysfunctional emotion regulation strategies and the reported experience of psychosomatic complaints such as headaches, stomach-ache, back-ache, feeling low, irritability, feeling nervous, sleep problems and dizziness. Overall, the frequent use of functional emotion regulation strategies was associated with a better subjective quality of life, whereas the frequent use of dysfunctional strategies was associated with a poorer subjective quality of life.

In relation to specific emotions, Phillips (2003) study supported the idea that certain strategies are used more often with certain emotions (Diener & Mangelsdorf, 1999).

The relationship between the use of functional and dysfunctional emotion regulation strategies and physical complaints will be further explored in this study, focusing specifically on primary chronic headache. To the authors knowledge, no research to date has attempted to identify the emotion regulation strategies of chronic headache sufferers. It is hoped that this study will provide an insight into this issue, and generate questions that will elicit further research in this field. The next section aims to integrate the issues discussed so far by making a link between pain and emotion in the light of the literature available.

1.4.6. Summary

The notion of emotion regulation as a process inherent to human nature, as well as an important factor in mental and physical health, is well established in the literature. Systematic research on the field of emotion regulation, however, is relatively new. The literature suggests that individual differences in emotion regulation strategies appear to be a strong indicator of how emotion regulation styles affect psychological and physical well being. Accordingly, functional and dysfunctional emotional regulatory strategies, drawing on internal and external resources, have been identified and linked to different outcomes in psychopathology and psychosomatic complaints. Overall, the evidence suggests that the frequent use of functional strategies is related to better outcomes.

1.5. Pain and Emotions

In order to understand pain and associated emotions it is important to be able to measure it. However, as a multidimensional experience, pain possesses a number of qualities varying over a wide range of intensities, rendering quantification a very difficult task. It has been argued that one of the difficulties associated with this is the fact that far too many words are used to describe pain and emotions, calling for a smaller number of dimensions or clusters (Clark, Janal & Carroll, 1989).

1.5.1. Dimensions of Pain and Emotion

Various dimensions of pain and emotion have been proposed. Melzack (1973) noted that while Aristotle viewed pain as an emotion, rather than a sensation, physiologists in the nineteenth century conceived pain as a sensory modality devoid of emotion. Later these views were combined by Sherrington (1900) who proposed that pain had two dimensions: *sensory* and *affective*. The affective tone was seen as an attribute of all sensations.

Melzack and Casey (1968) argued for a three-dimensional conceptual model including a *sensory-discriminative* dimension, an *affective-motivational* dimension and a *cognitive-evaluative* dimension. The sensory-discriminative dimension includes intensity, temporal and spatial properties as well as somatosensory qualities of pain. The affective-motivational dimension concerns the emotional and aversive aspects of pain and suffering. The cognitive-evaluative dimension concerns the individual's evaluation of the meaning and possible consequences of the pain, including its impact on quality of life and the possibility of death. This model is still widely accepted and has been incorporated into the definition of pain presented by the

International Association for the Study of Pain (1986), as it integrates the physiological and psychological aspects of pain and suffering.

Building on Melzack and Casey's model, Loeser (1980) and Fordyce (1988) have described four aspects of pain and suffering: *nociception* – the nerve fibre excitation resulting from tissue-damaging stimuli; *perception of pain* – the sensation that makes the person aware of the pain which may or may not follow nociceptive stimulation; *suffering* – the negative affective and emotional responses such as anxiety and depression; and *pain behaviours* – the observable behaviours of individuals experiencing pain, including facial and verbal expressions, resting or taking medication.

1.5.2. Multidimensional Scaling

The Melzack-Casey and Loeser-Fordyce models are complementary and widely accepted, but more recent research on multidimensional scaling indicates that there may be other dimensions (e.g. Fernandez & Turk, 1992; Clark, Janal, Hoben & Carroll, 2001; Janal, 1995). Multidimensional scaling is an objective way of investigating dimensions of pain and suffering and has contributed considerably to advances in the understanding of pain and emotion. It is based on the view that pain and emotion not only can be represented by specific dimensions, but also that different individuals can be placed at different points along these dimensions (Janal, 1995). However, it has been argued that obtaining a pure measure of each dimension is a rather difficult task requiring innovative research methods (Fernandes & Turk, 1992).

Some researchers have questioned the separability of the sensory and affective dimensions (e.g. Fernandez & Turk, 1992; Merskey & Spear, 1967). Mersey and Spear (1967) argued that the separation between sensation and pain perception could be merely semantic, and that it might be more appropriate to conceive these as contributory causes rather than separable components of pain. Fernandez and Turk (1992) argued that although there is research evidence supporting the separability of the sensory and emotional components of pain, these findings are ambiguous mostly because separation does not necessarily imply independence, as there is often a covariance between sensory and affective variables in pain.

1.5.3. Anger and Pain

According to the gate control theory, anger is one of the negative emotions that can increase pain intensity by altering descending and central pain modulation systems (Melzack, 1999). Individuals suffering from conditions characterized by persistent pain often report feeling angry (e.g. Zimmerman, Story, Gaston-Johasson & Rowles, 1996) and these angry feelings can be targeted at themselves or others (Okifuji, Turk & Curran, 1999).

1.5.3.1. Anger Constructs

A recent review on anger and persistent pain (Greenwood, Thurston, Rumble, Waters & Keefe, 2003) noted that studies investigating the relationship between anger and pain have focused on distinct but related constructs namely anger, hostility and aggression. Although closely related, these constructs are said to differ regarding their emotional, cognitive and behavioural components. Anger has been conceptualised as a transitory state occurring in response to perceived unfair treatment

or harm (Berkowitz, 1990; Tomkins, 1991). While an angry emotional reaction can be adaptive, chronic angry emotional reactions are seen as maladaptive as they may lead to pervasive interpersonal disruption and induce chronic sympathetic activation (Greenwood, Thurston, Rumble, Waters & Keefe, 2003). Individuals who experience anger more frequently than others are described as being high in trait anger (Deffenbacher, 1992).

The term hostility describes an enduring tendency to make cognitive appraisals of malicious intent of others or anticipation of mistreatment from others (Smith, 1992). Hostile individuals are described as having the traits of cynicism, distrust and anger (Friedman, 1992).

Aggression refers to punitive or destructive behaviours including physical assaults and verbal attacks (Spielberger, 1988). This anger related construct is not usually measured in pain studies, however its relevance is often implied since aggressive behaviour often represents a pathway by which hostility or anger is manifested (Greenwood, Thurston, Rumble, Waters & Keefe, 2003).

1.5.3.2. Dimensions of Anger

Some studies have reported high levels of anger in chronic pain patients in clinical settings (e.g. Braha & Catchlove, 1986; Bruehl, Burns, Chung, Ward & Johnson, 2002; Burns, Johnson, Devine, Mahoney & Pawl, 1998; Kinder & Curtiss, 1988; Schwartz, Slater, Birchler & Atkinson, 1991). Generally, two important dimensions have been described and investigated in the research involving chronic pain patients. The first dimension concerns state versus trait anger. Spielberger, Jacobs, Russel &

Crane (1983) defined state anger as a transitory emotional phase, whereas trait anger is seen as a relatively enduring predisposition to experience the emotion of anger. The other dimension concerns anger expression, which includes the concepts of 'anger-in' and 'anger-out'. Anger-in refers to the degree to which emotional feelings are suppressed and anger-out concerns the degree to which angry feelings are expressed through aggressive acts (Spielberger, 1988). Another important concept is that of 'passive aggressiveness' which refers to the behavioural communication of anger expressed by non-cooperation rather than overt aggression (Fernandez & Turk, 1995).

Inbuilt in the concept of anger-in is the notion that that individuals who do not express their anger outwardly, experience considerable internal stress. Furthermore, it has been suggested that failure to express anger, combined with a lack of awareness that anger is being held in, may result in the experience of somatic symptoms (Eckhardt & Deffenbacher, 1995). In fact, research has demonstrated that high levels of anger-in have a substantial negative impact on physical health (e.g. Fernandez & Turk, 1995; Pennebaker, 1989; Suinn, 2001).

Research investigating the relationship between anger and pain has shown that suppressed anger is strongly associated with chronic pain. For instance, it has been found that chronic pain sufferers are more likely to suppress their anger than pain-free controls (e.g. Arena, Bruno, Rozantine & Meador, 1997; Braha & Catchlove, 1986; Nicholson, Gramling, Ong & Buenevar, 2003). Moreover, an individual's failure to express anger has been associated with higher pain frequency, intensity and interference (Burns, Weigner, Derleth, Kiselica & Pawl, 1997; Kerns, Rosenberg &

Jacob, 1994). In addition, inhibition of angry feelings has been found to be an important element of the affective component of pain (Fernandez & Milburn, 1994; Wade, Price, Hamer, Schwartz & Hart, 1990).

Higher levels of trait anger and hostility have also been identified in chronic pain patients (e.g. Kinder, Curtiss & Kalichman, 1986; Schmidt & Wallace, 1982). This is the case despite findings that low back pain and headache patients have a tendency to deny feelings of anger and aggressiveness, in comparison with pain-free control subjects (Franz, Paul, Bautz, Choroba & Hildebrandt, 1986). Regarding the relationship between pain and trait anger, it has been suggested that high levels of trait anger may lead to an increase in skeletal muscle tension resulting in pain (Burns, 1997).

1.5.3.3. General Implications

The angry states (overt anger or inhibited anger) experienced by chronic pain sufferers are known to have widespread consequences. First, it is likely to affect interpersonal relationships. For instance, Schwartz, Slater, Birchler & Atkinson (1991) found that depression and marital dissatisfaction amongst spouses of chronic pain patients was attributable to patient's pain, anger and hostility. Second, it has been suggested that the internalisation and externalisation of anger has implications for physical well being and is particularly linked to hypertension and coronary heart disease (Diamond, 1982). According to Fernandez and Turk (1995), anger associated with chronic pain, unless appropriately expressed and regulated, could be a potential threat to cardiovascular health. Third, it has been argued that hostility is associated with poor health habits (Schweritz & Rugulies, 1992). Fernandez and Turk (1995)

pointed out that those chronic pain patients who develop hostility may be at risk of adopting a maladaptive lifestyle that generates additional health problems.

There is evidence that anger can affect the treatment outcome of chronic pain patients. For example, it has been found that patients with higher levels of anger suppression show less improvement following cognitive behavioural intervention for pain (Burns, Johnson, Devine, Mahoney & Pawl, 1998). Based on their clinical experience, Fernandez and Turk (1995) suggested that pain patients' manifestation of anger (such as hostility, cynicism and mistrustfulness) could undermine the therapeutic relationship and the attainment of treatment goals. Furthermore, they suggested that angry pain patients may potentially disrupt the group process usually used in pain management programmes. One example cited is the damage that may be caused by other group members modelling the uncooperative and hostile behaviour of angry group members.

1.5.4. Summary

Various dimensions of pain and emotion have been proposed. While most models distinguished between sensory, affective and cognitive dimensions, the separability between the sensory and affective dimensions has been questioned, as there is often a covariance between sensory and affective variables.

Anger is one of the most prominent emotions associated with pain. High levels of trait anger and anger-in have been identified in chronic pain patients, including headache sufferers, in comparison with pain-free individuals. It has been suggested that anger suppression has a substantial negative impact on chronic pain. In addition,

research has demonstrated that the angry states of chronic pain patients have an impact on their interpersonal relationships and on treatment outcome.

1.6. The Headache Phenomenon

The Classification Subcommittee of the International Headache Society has recently reviewed its 1988 hierarchical system to classify and diagnose headache disorders for research and clinical practice (International Headache Society, 2004). The term 'headache' is used broadly to describe a variety of headache types. Headache disorders are classified into primary (idiopathic) and secondary (symptomatic). Primary headaches occur spontaneously without an apparent organic cause and are classified into chronic or episodic.

Various types of primary headaches have been distinguished (cf. International Headache Society, 1988, 2004; Silberstein, Lipton & Sliwinski, 1996): e.g. migraine, tension-type headache, cluster headache, chronic daily headache and other miscellaneous headaches. It is common for patients to experience more than one type of headaches (e.g. combined migraine/tension-type), and they are able to distinguish between them (Laughey, MacGregor & Wilkinson, 1993).

Secondary headaches result from another underlying organic cause, such as tissue damage, and encompass a range of conditions including structural lesions or trauma in the head area and adverse reactions to drugs or medical procedures. The mechanisms for primary headaches, however, are less well understood and cannot be directly associated with tissue damage or nociception. The most common types of chronic headache are migraine and tension-type headache (Martin, 1993). These will now be discussed in more detail.

1.6.1. Migraine

Migraine is a common primary headache disorder which has now been ranked by the World Health Organization as number 19 amongst all diseases in the world that cause disability (International Headache Society, 2004). Physiologically, migraine has nervous system and vascular mechanisms, and possibly some genetic component (Martin, 1993). Increased trigeminal ganglion activity has been observed during migraine attacks and it has been suggested that nitric oxide is one of the substances that induces migraine (Edvinsson, 2001). Migraine has also been associated with cortical spreading depression, which is a short-lived, reversible depression of electrical activity that moves from the rear to the front of the brain, which may affect the blood flow and activate the meningeal trigeminal nerve fibres (Lauritzen, 2001; Olesen, Tfelt-Hansen & Welch, 2000).

According to the International Headache Society (2004), migraine can be divided into two major sub-types: *migraine without aura* and *migraine with aura.* Migraine without aura is a recurrent headache disorder which manifests in attacks that last 4-72 hours. It is described as a headache of unilateral location and pulsating (i.e. throbbing) quality. Its intensity can be moderate or severe and may be aggravated by physical activity. It is also associated with nausea, vomiting, photophobia and phonophobia. Migraine without aura is the most common type of migraine, having a higher average attack frequency. Very frequent attacks are now distinguished as chronic migraine. It is also regarded as more disabling than migraine with aura. In addition, migraine without aura seems to be related to menstruation.

Migraine with aura manifests in attacks of reversible focal neurological symptoms that precede and may also accompany the headache. Common visual symptoms are flickering lights, spots or lines, and loss of vision. Sensory symptoms include pins and needles and numbness. Fully reversible dysphasic speech disturbance may also occur. These symptoms develop gradually over 5-20 minutes and often last for less than 60 minutes (International Headache Society, 2004).

Premonitory symptoms may also occur hours to a day before a migraine attack (with or without aura). These may include fatigue, yawning, concentration difficulties, neck stiffness, sensitivity to light or sound, blurred vision, nausea and paleness (International Headache Society, 2004).

1.6.2. Tension-Type Headache

Tension-type headache is the most common type of primary headache and it is divided into two types: episodic and chronic (International Headache Society, 2004; Jensen, 1999; Martin, 1993). The physiological mechanisms of tension-type headache have been a matter of debate for decades (Jensen, 1999; Martin, 1993). The key issue is whether tension-type headache originates from miofascial tissues or from central mechanisms in the brain. Historically, prior to 1988, when then International Headache Society published a hierarchical system to classify headache disorders (International Headache Society, 1988), this headache syndrome was called muscle contraction headache. It was then believed that the underlying causes of this type of headache was tight and spastic muscles in the shoulders, neck and face (Martin, 1993). However, it is now thought that sensitisation of peripheral nerves in

contracted shoulder, neck and facial muscles as well as sensitisation of the central nervous system are both involved in tension-type headache (Jensen, 2001).

In the most recent classification by the International Headache Society (2004) the episodic subtype has been further subdivided into *infrequent* (i.e. less than once per month) and *frequent* (i.e. more than 1 but less than 15 days per month), lasting from 30 minutes to 7 days. The infrequent subtype does not have much impact on the individual and in general is not seen as a cause of concern from the medical point of view. The frequent sufferers, however, usually encounter considerable disability warranting more attention from the medical profession. Both the frequent and infrequent subtypes are described as typically bilateral, pressing or tightening in quality and of mild to moderate intensity. Physical activity does not make it worse and there is no nausea, although photophobia or phonophobia may be present. The chronic subtype headaches (i.e. occurring on more than 15 days per month) last hours or may be continuous. Its descriptive qualities are similar to the episodic tension-type headaches except that mild nausea may be present. Chronic tension-type headache is a serious disease which has a profound impact on the individual, as it is associated with considerable disability, decrease in quality of life and high personal and socio-economic costs (International Headache Society, 2004; Martin, 1993).

1.6.3. Epidemiology

Studies have indicated that most people experience headaches during their lifetime with estimated lifetime prevalence ranging from 68% to 96% (e.g. Rasmussen, Jensen, Schroll & Olesen, 1991; Ziegler, Hassanein & Couch, 1977) and yearly prevalence varying between 40% and around 90% in different studies (e.g. Linet,

Stewart, Celentano, Ziegler & Sprecher, 1989; Prencipe, Casini, Ferretti, Santini, Pezella, Scaldaferr & Culasso, 2001). Population studies conducted in the UK have estimated that more than two-thirds of the adult population will suffer from headaches in a one year period, however, these studies were carried out around thirty years ago (e.g. Clarke & Waters, 1974; Waters, 1970).

Regarding age stratification, these studies indicated that there is a headache prevalence peak during the twenties and early thirties and a decline in old age. However, not many studies have looked at how headaches change over time and results are mixed. For instance, Pearce (1993) found that patients suffering from cluster headaches described the same pattern of headaches after 10 to 25 years. Similarly, a recent large study found that estimates of migraine prevalence remained unchanged after 10 years (Lipton, Stewart, Diamond, Diamond & Reed, 2001). A study carried out amongst over 65 year old headache sufferers found that, compared with 10 years previously, headaches remained unchanged for two-thirds of them, while one quarter reported improvement (Wang, Liu, Fuh, Liu, Lin, Chen, Lin, Wang, Hsu, Wang & Lin, 1997). A longitudinal study following children with migraine aged 7 to 13 over a 40 year period found that almost one-third reported migraine every year during that period, while one-fifth still suffered from migraine but had experienced migraine-free years during follow-up, and the remainder was no longer suffering from migraine (Bille, 1997).

Headache prevalence has also been associated with sociodemographic factors. In relation to gender, a female preponderance has been found for both migraine and tension-type headache with male:female ratio being around 1:3 for migraine and 4:5

for tension type headache (Rasmussen, 1995). According to the International Headache Society (2004), for cluster headache the prevalence is 3-4 times higher in men than in women.

Some studies in the United States have found an association between headaches and educational level. The prevalence of tension-type headache and migraine was found to be higher amongst those educated to a higher level (Schwartz, Stewart, Simon & Lipton, 1998; Stang & Osterhaus, 1993). In relation to income, while a Canadian study found that both tension-type headache and migraine were unrelated to income (Pryse-Phillips, Findlay, Tugwell, Edmeads, Murray & Nelson, 1992), an American study found higher prevalences of migraine amongst those with lower incomes (Stewart, Lipton, Celentano & Reed, 1992). More recently, a large Norwegian study found that low social class, low income and fewer years of education were associated with increased risk of chronic headache (Hagen, Vatten, Stovener, Zwart, Krokstad & Bovim, 2002).

Estimated headache prevalence seems to vary across the world. Studies in North America and Europe (e.g. Linet, Stewart, Celentano, Ziegler & Sprecher, 1989; Rasmussen, Jensen, Schroll & Olesen, 1991; Newland, Illis, Robinson, Batchelor & Waters, 1978) have found higher prevalences in comparison with African and Asian studies (e.g. Sakai & Igarashi, 1997; Wang, Fuh, Young, Lu & Shia, 2001; Roh, Kim & Ahn, 1998). It is not clear whether these differences are due to genetic, environmental or cultural factors.

1.6.4. Headache-related Disability

Headache is a common phenomenon familiar to most people and often regarded as a minor ailment. However, the impact of recurrent headaches cannot be underestimated and many headache sufferers experience considerable disability. In this context, disability represents any restriction or inability to perform activities in a way or within the range that is considered normal (World Health Organization, 1980). Disability assessment often include examining patients' emotional response to impairment, their ability to engage in necessary daily activities and their ability to interact with others in a variety of settings. Accordingly, successful treatment intervention could be indicated by a reduction in self-perceived disability (Jacobson, Ramadan, Aggarwal, Craig & Newman, 1994).

Different types of primary headaches present differing disability levels. In relation to the socio-economic impact of headaches, represented by reduced capability and absence from work, it has been demonstrated that migraine causes more disability than other types of severe headaches, with estimates from different studies ranging from 25% to 78% of migraine sufferers reporting that their headache affected their work, compared with only 8% to 38% of tension-type headache sufferers (Cull, Wells & Miocevich, 1992; Dowson & Jagger, 1999; Edmeads, Findlay, Tugwell, Pryse-Phillips, Nelson & Murray, 1993; Mounstephen & Harrison, 1995; Pryse-Phillips, Findlay, Tugwell, Edmeads, Murray & Nelson, 1992). However, it has been argued that, even though tension-type headache sufferers have reported lower levels of disability than migraine sufferers, the fact that there is a higher prevalence of this condition means that tension-type headache has a greater impact in the population as whole (Rasmussen, 1995). In fact, migraine sufferers not only report having missed

days at work due to their migraines but also due to other types of headache (Von Korff, Stewart, Simon & Lipton, 1998). Of those who continue to work during their headaches, migraine sufferers reported a decrease of 50% to 60% in effectiveness while other type of headache sufferers reported a smaller reduction in effectiveness of around 24% to 28% (Von Korff, Stewart, Simon & Lipton, 1998).

Regarding tension-type headache, it has been found that the number of days affected was higher for those suffering from chronic as opposed to episodic tension-type headache, with 8% of episodic tension-type headache sufferers reporting having missed an average of 9 working days and 44% reporting having had an average of five reduced effectiveness days. In comparison, around 11.8% of those suffering form chronic tension-type headaches reported having missed an average of 27 working days with 47% describing an average of 20 reduced effectiveness days (Schwartz, Stewart, Simon & Lipton, 1998).

It has been shown that chronic headache also affect the family life and social activities of sufferers. Studies have demonstrated that headaches have an adverse affect on headache sufferers' relationships with family and friends, as they often need to curtail family and social activities (Edmeads, Findlay, Tugwell, Pryse-Phillips, Nelson & Murray, 1993; Lipton, Stewart, Diamond, Diamond & Reed, 2001; Pryse-Phillips, Findlay, Tugwell, Edmeads, Murray & Nelson, 1992). In addition, it has been found that around 40% of headache sufferers worried about the occurrence of headache during a social event and 45% had concerns about driving with a headache (Edmeads, Findlay, Tugwell, Pryse-Phillips, Nelson & Murray, 1993).

The detrimental effect of chronic headache in the overall health-related quality of life of headache sufferers is well documented. Similar patterns have been found for migraine, tension-type headache and mixed headache, with role functioning and social functioning representing the areas of greatest reduction in quality of life (Essink-Bot, van Royen, Krabbe, Bonsel & Rutten, 1995; Osterhaus, Townsend, Gandek & Ware, 1994).

Although the above mentioned studies have shown that headache sufferers experience increased levels of disability and reduced quality of life it is not clear what aspects of the headache are associated with these effects. As with other types of chronic pain, headache disorders are highly variable in terms of frequency and intensity with patients varying considerably in the way they respond to pain (Henry, Auray, Guadin, Dartigues, Duru, Lanteri-Minet Lucas, Pradalier, Chazot & El Hasnaoui, 2002; Lipton, Scher, Kolodner, Liberman, Steiner & Stewart, 2002). A recent study investigating the clinical features that have the greatest impact on migraine sufferers' quality of life, found that headache intensity, rather than frequency, is a major determinant of self-reported headache-related physical and emotional disability (Magnusson & Becker, 2003). These findings were consistent with previous studies where the intensity of the headache was found to be a more powerful predictor of the severity of psychological symptoms than the frequency (e.g. Jacobson, Ramadan, Aggarwal & Newman, 1994; Scharff, Turk & Marcus, 1995).

1.6.5. Psychophysiology of Headaches

Over the last few decades research on headache disorders, especially migraine and tension-type headaches, has altered the general understanding of the psychological

component of these disorders. Still, no specific patterns of psychopathology for either migraine or tension-type headaches have yet been identified (Donias, Peioglou-Harmoussi, Georgiadis & Manos, 1991; Martin, 1993). Nevertheless, most clinicians and researchers agree that these disorders are not purely or primarily physical and the role of psychopathological mechanisms in the headache process has received considerable attention, especially regarding predisposing, precipitating and maintaining factors (Donias, Peioglou-Harmoussi, Georgiadis & Manos, 1991; Martin, 1993).

1.6.5.1. Personality Factors

The view that headache sufferers have a particular personality profile has been argued for years. For instance, the association between unexpressed anger and migraine can be found in writings dating as far back as 240 years ago (Harrison, 1975). Early investigations based on uncontrolled clinical interviews described migraine sufferers as tense, ambitious, perfectionists, compulsive and orderly, inflexible, resentful and unable to express aggressive feelings constructively (Alvarez, 1974; Friedman, von Storch & Merritt, 1954; Wolff, 1937). Descriptions of the personality traits associated with tension-type headache bare some resemblance with those of migraineurs. For example, they have been described as sensitive, depressed, perfectionist, worrisome, chronically tense, apprehensive, hostile, dependent and psychosexually conflicted (Martin, 1966; Martin, Rome & Swenson, 1967).

An extensive review by Blanchard, Andrasik & Arena (1984) of the literature on personality and headache, indicated that findings are rather equivocal and do not support the notion of a 'headache personality'. However, based on this literature they

suggested that headache sufferers in general are more psychologically distressed and present more deviant personality characteristics than nonheadache sufferers, with tension-type headache sufferers presenting more discrepancies on most measures.

Since then, studies investigating the psychological profile of headache sufferers continued to present contradictory results. Ziegler & Paolo (1995) found that headache sufferers scored significantly higher than nonheadache controls on the Minnesota Multiphasic Personality Inventory (MMPI) and that these psychological characteristics are important factors in the decision to seek medical help. Using the Freiburgh Personality Inventory (FPI), Merikangas, Stevens & Angst (1993) found that migraneurs showed elevated rates of neuroticism compared to nonheadache controls. The same study found that tension-type headache sufferers did not differ from controls on any of the personality factors.

Studies examining whether the psychological profile differ according to headache type yielded mixed results. While recent research suggested that headache groups differ regarding personality and psychopathological profiles (e.g. Bigal, Sheftell, Rapoport, Tepper, Weeks & Baskin, 2003), other studies concluded that it is not possible to distinguish between headache diagnostic groups (Pfaffenrath, Hummelsberger, Pollmann, Kaube & Rath, 1991; Robinson, Geisser, Dieter & Swerdlow, 1991). In fact, it has been proposed that the MMPI scale types may reflect a patient's response to pain and are therefore more likely to be the result of coping resources than headache-related personality style (Robinson, Geisser, Dieter & Swerdlow, 1991).

Inspired by the similarities between Type A behaviour pattern and personality descriptions of migraineurs, some studies have investigated the relationship between headaches and Type A personality. Some research indicated that Type A individuals experienced significantly more tension headaches and migraines than did Type B individuals (Hicks & Campbell, 1983; Martin, Nathan & Milech, 1987; Morgan, Day, Jefferson & Harris, 1984). It has also been found that the majority of chronic headache sufferers displayed the Type A behaviour pattern (Martin, Nathan & Milech, 1987). However, two studies have suggested that the relationship between headaches and Type A behaviour pattern is much weaker than expected (Hillhouse, Blanchard, Applebaum & Kirsch, 1988; Rappaport, McAnulty & Brantley, 1988).

One important question in the literature is whether or not personality factors play an specific role in the etiology of headaches. A further question is whether distinct profiles might predict outcome. It has been suggested that psychopathology may function either as a cause or a consequence of headaches (e.g. Collet, Cottraux & Juenet, 1986). Research examining changes in personality functioning as a result of treatment is inconclusive. Some studies showed that traits such as neuroticism and hysteria were still present after successful treatment (e.g. Mongini, Defilippi & Negro, 1997; Sovak, Kunzel, Sternbach & Dalessio, 1981), lending support to the idea that personality traits are not a result of the pain experience (Arena, Andrasik & Blanchard, 1985). It has been argued that personality traits could be a predisposing factor for headaches precipitated by stress but not for headaches precipitated by perceptual stimuli (Martin, 1993). More recently, studies have proposed that depression has a strong effect on elevated personality trait scores and is likely to influence pathophysiology and treatment outcome in the long term (Mongini, Keller,

Deregibus, Raviola, Mongini & Sancarlo, 2003; Boz, Sayar, Velioglu, Hocaoglu, Alioglu, Yalman & Ozmenoglu, 2004).

1.6.5.2. Stress and Emotions

Stress is one of the precipitating factors most frequently reported by headache sufferers, occurring with similar frequency in migraine and tension-type headaches (Dowson & Jagger, 1999; Martin, 1993; Ulrich, Russel, Jensen & Olesen, 1996).

A recent study on life event stress and headache found that while positive life stress was not associated with headache frequency, negative life stress was related with a high headache frequency especially in women (Reynolds & Hovanitz, 2000). However, it has been previously argued that the psychological context of the event is more important than the event itself (Levor, Cohen, McArthur & Heuser, 1986) and it has been suggested that there might be a mental or cognitive 'screening' of the emotional experiences that are associated with a headache attack. This cognitive screening involves an awareness of vulnerability to specific emotional precipitants of headaches (Donias, Peioglou-Harmoussi, Georgiadis & Manos, 1991).

A study investigating whether migraine and tension-type headache sufferers differed regarding emotional states perceived as precipitants of their headaches, also examined whether they differed in their awareness of vulnerability to different emotional precipitants (Donias, Peioglou-Harmoussi, Georgiadis & Manos, 1991). Overall, results indicated that the most common precipitating factors in tension-type headache involved negative emotional arousal. In this sample, tension-type headache sufferers associated anger and anxiety with an attack more frequently than migraine sufferers,

who indicated that any emotional arousal, irrespective of its nature (i.e. including positive states such as feeling happy and excited) and frequency, seemed to precipitate an attack. Results also demonstrated that tension-type headache and migraine patients differed regarding the awareness of vulnerability to different emotional precipitants. This was due to migraneurs being more frequently aware of positive states precipitating an attack of migraine.

The authors postulated that distinct cognitive shemata both in tension-type headache and migraine function either over-effectively (as screening mechanisms processing emotional arousal information in an inflexible way) or defectively (based on learned vulnerabilities to the specific emotional information). It was suggested that over-effective schemata assessing any emotional arousal as a dangerous signal (in migraine), or selectively assessing negative emotional arousal as signals for alertness or even counterattack (in tension-type headache), could trigger off the physiological mechanisms resulting in the experience of pain, almost as a protective mechanism whereby pain alerts the organism of potential danger.

While this model seem to over-emphasise the cognitive aspect in the precipitation process of migraine and tension-type headaches, the authors stressed that the emotional variables involved are just as important, as they provide the necessary instrument for the cognitive mediation (Donias, Peioglou-Harmoussi, Georgiadis & Manos, 1991).

1.6.5.3. Anger and Headaches

As described above, earlier investigations into the personality characteristics of chronic headache sufferers have described them as angry, hostile and tense individuals (Friedman, Van Storch & Merritt, 1964; Martin, 1966; Wolff, 1937). However, it has been pointed out that these conclusions were only implied by the data and therefore more direct and systematic measurement of anger in this group of patients was needed (Arena, Blanhard & Andrasik, 1984; Fernandez & Turk, 1995).

More recently, a few studies have been conducted to systematically investigate the relationship between anger and headache. Studies comparing headache sufferers with headache-free individuals have demonstrated that the former have significantly higher levels of trait anger and anger-in (Arena, Bruno, Rozantine & Meador, 1997; Hatch, Schoenfeld, Boutros, Seleshi, Moore & Cyr-Provost, 1991; Materazzo, Cathcart & Pritchard, 2000) and that levels of anger-in exceeded those for anger-out in chronic headache sufferers (Tschannen, Duckro, Margolis & Tomazic, 1992).

Some studies suggested that anger and hostility have an etiological role in chronic headache (Braha & Catchlove, 1986; Hatch, Schoenfeld, Boutros, Seleshi, Moore & Cyr-Provost, 1991). However, it has been argued that since these studies were only correlational, it may be less contentious to suggest that the suppression of anger has an exacerbating influence rather than a precipitating effect on pain (Fernandez & Turk, 1995).

While these studies have provided a more empirically based understanding of the anger-headache relationship, the fact that other forms of negative affect (e.g. anxiety

and depression) have been shown to be related to headaches indicate that it is paramount to investigate whether the anger-headache relationship stands after controlling for depression and anxiety. In fact, two studies investigating whether anger and depression predicted headache-related disability showed that anger-in only affected disability indirectly, via depression (Duckro, Chibnall & Tomasic, 1995; Tschannen, Duckro, Margolis & Tomazic, 1992).

A recent study carried out in a non-clinical population, proposed to examine whether anger and anger expression differed between headache sufferers and headache-free individuals after controlling for depression and anxiety (Nicholson, Gramling, Ong & Buenevar, 2003). Results indicated that headache sufferers held their anger in significantly more than headache-free individuals, even after controlling for levels of trait anger, depression and anxiety. Furthermore, anger-in was found to be the only significant predictor of headache, after controlling for trait variables (e.g. depression, anxiety, hostility and trait anger).

The authors pointed out that, while these results provided further evidence that depression, anxiety and anger are interrelated and prominent amongst headache sufferers, further research is needed to ensure that these findings generalise to a clinical population of headache sufferers. As such, one of the aims of this study is to investigate the prominence of anger in a clinical population of headache sufferers in comparison with headache-free controls. In addition, it is also proposed to investigate whether anger-related variables contribute to predicting severity of headache and headache-related disability beyond that accounted for by depression and anxiety.

1.6.5.4. Other Precipitating Factors

Studies investigating other precipitators of headache attacks have yielded mixed results. Migraine sufferers have reported lack of sleep, menstruation, food, alcohol and sun as triggers (Dowson & Jagger, 1999). The relationship of headache with alcohol is not clear though and one study found that alcohol consumption was not associated with either migraine or tension-type headache (Rasmussen, 1993). The same study found that smoking was not associated with headache occurrence and neither was caffeine intake, although there seemed to be a trend for lower prevalence in coffee drinkers. This study also showed that migraine was not associated with level of physical activity, although tension-type headache in men was more prevalent in the inactive group. It has been found that severely painful headaches are associated with the overuse of medication particularly for those with chronic conditions (Castillo, Munoz, Guitera & Pascual, 1999; Phillips, 1977; Prencipe, Casini, Ferretti, Santini, Pezella, Scaldaferri & Culasso, 2001).

1.6.6. Coping Mechanisms of Headache Sufferers

A number of studies have demonstrated that coping styles affect patient's adjustment to pain (e.g. Endler, Corace, Summerfeldt, Johnson & Rothbart, 2003; Keefe, Crisson, Urban & Williams, 1990; Turner, Jensen & Romano, 2000) and therefore psychological treatments for chronic pain usually aim to modify maladaptive coping strategies (Keefe, Dunsmore & Burnett, 1992; ter Kuile, Spinhoven, Linssen & van Houwelingen, 1995).

Certain dispositional coping styles have been found to be associated with pain and distress. For instance, passive coping strategies (e.g. 'hoping' and 'praying') and

emotion-focussed strategies (e.g. catastrophising, emotional preoccupation coping) have been shown to be positively related to perceived pain severity and emotional distress such as depression and anxiety (e.g. Endler, Corace, Summerfeldt, Johnson & Rothbart, 2003; Gil, Williams, Keefe & Beckham, 1990; Jensen, Turner, Romano & Karoly, 1991; Keefe, Brown, Wallston & Caldwell, 1989; Rosenstiel & Keefe, 1983). In contrast, it has been shown that chronic pain sufferers who employ action-oriented coping strategies (e.g. seeking information and medical advice) have lower levels of depression in comparison with those who do not use this type of strategy (Spinhoven, ter Kuile, Linssen & Gazendam, 1989).

The long-term use of avoidant coping strategies has been linked to pain severity, emotional distress, decreased social activity and functional impairment including loss of employment (Holmes & Stevenson, 1990; Katz, Ritvo, Irvine & Jackson, 1996; Summerfeldt & Endler, 1998). In contrast, it has been found that chronic pain sufferers who employ attentional coping strategies (e.g. distraction) have lower levels of depression, anxiety, pain severity and are more socially active (Holmes & Stevenson, 1990; Katz, Ritvo, Irvine & Jackson, 1996; Keefe & Williams, 1990).

A small number of studies have examined the coping strategies of headache sufferers (e.g. Ficek & Wittrock, 1995; Rollnik, Karst, Fink & Dengler, 2001; ter Kuile, Spinhoven, Linssen & van Houwelingen, 1995; Spinhoven, Jochems, Linssen & Bogaards, 1991). A review of several controlled studies indicated that direct attempts to change pain-related thoughts and cognitive coping strategies contributed to reducing headache severity (Bogaards & ter Kuile, 1994). Interestingly, one study found that patients with episodic tension-type headache did not differ from healthy

controls in terms of pain-coping strategies, despite presenting higher levels of depression and trait anxiety (Ficek & Wittrock, 1995). In contrast, those suffering from chronic tension-type headache have been shown to have lower level of active coping with pain (Spinhoven, Jochems, Linssen & Bogaards, 1991).

A study comparing the coping strategies of episodic and chronic tension-type headache found that disadvantageous coping with illness strategies (e.g. avoidance behaviour, endurance strategies) might contribute to the transformation from episodic into chronic tension-type headache (Rollnik, Karst, Fink & Dengler, 2001).

In relation to stressful events, assumptions that migraine sufferers are characterised by maladaptive or negative cognitive coping and cognitive evaluative processes, have received only partial support (Kroner-Herwig, Fritsche & Brauer, 1993).

1.6.7. Co-morbid Psychopathology

Co-morbid psychological conditions such as anxiety and depression appear to be high in migraine (Breslau, Merikangas & Bowden, 1994; Breslau, Schultz, Stewart, Lipton, Lucia & Welch, 2000; Mitsikostas & Thomas, 1999) and chronic tension-type headache (Holroyd, Stensland, Lipchik, Hill, O'Donnell & Cordingley, 2000; Marcus, 2000; Puca, Genco & Prudenzano, 1999). Patients with chronic daily headache have also been shown to present high levels of these conditions, with two-thirds having a depressive disorder, and one-third having an anxiety disorder (Juang, Wang, Fuh, Lu & Su, 2000). High levels of anxiety have also been found in those with cluster headache (Jorge, Leston, Arndt & Robinson, 1999).

It has been demonstrated that migraine sufferers are nearly three times more likely to suffer from depression when compared with controls (Lipton, Hamelsky, Kolodner, Steiner & Stewart, 2000). In migraine sufferers with depression, the negative effects on health status of having both conditions was greater than the additive affect of the individual conditions (Essink-Bot, van Royen, Krabbe, Bonsel & Rutten, 1995).

It appears that the association between migraine and depression is bi-directional: while migraine sufferers seem to be at increased risk of developing depression, current or previous depression seem to increase the risk of developing migraine. As for other severe non-migrainous headache this relationship seem to be unidirectional, with headache sufferers being at increased risk of developing depression (Breslau, Schultz, Stewart, Lipton, Lucia & Welch, 2000). This has important implications for the management of headaches, as it has been demonstrated that depressed headache sufferers have a poorer outcome on follow-up when compared with non-depressed

headache sufferers (Curioso, Young, Shechter & Kaiser, 1999; Devlen, 1994; Mitsikostas & Thomas, 1999). This highlights the importance of the identification and effective management of co-morbid disorders in the treatment of chronic headache (Holroyd, Stensland, Lipchik, Hill, O'Donnell & Cordingley, 2000).

1.6.8. Summary

The most common types of primary headache are migraine and tension-type headache, with a considerable number of patients experiencing both types. Epidemiological data indicate that chronic primary headache is a prevalent health problem worldwide affecting individuals across a wide age span. A considerable proportion of headache sufferers experience headache-related disability affecting work effectiveness, relationships, and health-related quality of life.

No specific patterns of psychopathology for headaches have yet been identified but it is believed that factors such as personality characteristics, stress and negative emotions such as anger, and coping styles may contribute to some extent to the maintenance of headache disorders. In addition, co-morbid disorders such as anxiety and depression are often present in headache sufferers.

1.7. Conclusions

The concept of pain encompasses sensory, cognitive and affective dimensions, involving complex interactions between biological, psychological and social factors. Pain is perceived as an emotional experience, where emotions are seen as fundamental to the experience of pain and not simply as a reaction to the sensation of pain.

Headache is generally perceived as a minor ailment affecting most individuals. Chronic headache, however, is a prevalent health problem and its effect on health and psychosocial functioning is well documented and cannot be underestimated. Research investigating the psychophysiology of chronic headache, especially migraine and tension-type headaches, have identified various factors that may contribute to the experience of headache attacks, including personality characteristics, stress, emotional states and coping styles.

As with other types of chronic pain, emotions play a significant role in the psychophysiology of headaches. In fact, negative emotions including anger, anxiety and depression are prominent amongst headache sufferers, and have been found to be predictive of headache severity and headache-related disability.

Research has indicated that emotion regulation styles affect psychological and physical well being. Given the central role of emotions in the experience of headaches, identifying the emotion regulation strategies frequently used by headache sufferers may prove essential for the development of interventions aimed at reducing

the frequency and severity of headache, consequently reducing its impact on patients' lives.

1.8. Aims of Study and Hypotheses

One of the aims of this study is to examine the emotions associated with the experience of chronic primary headache, by investigating the type of emotions that are prominent in this particular subgroup of chronic pain patients, in comparison with headache-free individuals, and the relationship between these emotions, pain severity and headache-related disability.

Another aim is to build on the scarce but growing literature examining the role of anger in chronic headache, and identify the relationship between anger and other affective states, such as depression and anxiety, with headache severity and headache-related disability.

This study also aims to identify how chronic headache sufferers regulate emotions and how this might differ from headache-free individuals. This aspect of the emotional component of pain has not yet been explored in this patient group and, therefore, it is hoped that this study will provide some insight into the strategies used by headache sufferers to regulate emotions. This might prove to be a potential area of research that could add to the current body of research on headache and chronic pain in general.

Based on the literature outlined above, it was hypothesised that:

1. Chronic headache patients will be found to experience the basic emotions of anger, fear and sadness more often than non-headache controls;

2. Chronic headache patients will show higher levels of trait anger and anger-in than non-headache controls.

3. Chronic headache patients will show higher levels of emotional disturbance (including anger, depression and anxiety) than non-headache controls;

4. There will be a positive correlation between the experience of negative emotions, pain severity and perceived disability, whereas a negative correlation will be found between the experience of positive emotions, pain severity and perceived disability;

5. There will be a positive correlation between emotional disturbance, pain severity and perceived disability;

6. There will be an association between the strategies used to regulate emotions, pain severity and perceived disability. Specifically, the frequent use of dysfunctional strategies will be positively correlated with pain severity and disability, whereas the frequent use of functional strategies will be negatively correlated with pain severity and disability;

7. The addition of anger-related variables to a model consisting of depression and anxiety will contribute significantly to a model predicting headache severity and headache-related disability.

2. Method

It was proposed to undertake a study to examine the emotional component of chronic headache and its effect on headache severity and headache-related disability. In considering this, the issues addressed included identifying basic emotions frequently experienced by chronic headache sufferers, level of emotional disturbance (e.g. anger, anxiety and depression) and styles of emotion regulation, in comparison with headache-free individuals. The protocol for this study was approved by the Lothian Research Ethics Committee, the Lothian Primary Care NHS Trust, and The Lothian University Hospitals NHS Trust (Appendix I).

2.1. Design

A cross-sectional mixed design was used for this research. This included a between subjects design to investigate the differences between headache and headache-free individuals, and a correlational design to examine the relationship between the measured variables.

2.2. Participants

A total of 146 individuals were invited to take part in this study. Out of these, 82 participants constituted the headache group. Participants were recruited from an outpatient Headache Clinic run at the Western General Hospital. Additionally, patients referred from the Headache Clinic to a pain management programme, based at the clinical psychology department of the Royal Edinburgh Hospital, were also invited to take part in the study, when they first attended for a psychological assessment. Participants were included in this group if they had been diagnosed by the clinic specialist as having a primary headache disorder (e.g. migraine, tension-type

headache, cluster headache, etc.) of a chronic nature, according to the International Headache Society criteria (1988, 2004). Exclusion criteria included evidence of tumour, recent substance abuse, systemic disease, history of head trauma and being aged under 18. Following these criteria, two participants were excluded, one of whom suffered from post-traumatic headache, and one other who was 16 years old. In addition, 3 patients refused to take part in the study and an additional 20 did not return their questionnaires. As a result, the headache group was reduced to a total of 57 participants.

The headache-free group constituted an opportunistic sample of 64 individuals, comprising acquaintances and colleagues of the author, as well as of other collaborators, including the clinical supervisor, and friends of the author. Exclusion criteria included experience of frequent headaches and of major medical or neurological disorders, and being aged under 18. Out of these, one participant was excluded due to suffering from migraine, and an additional 16 did not return their questionnaires. This reduced the headache-free group to a total of 47 participants.

Overall, 104 individuals took part in this study. An attempt was made to match controls to headache participants on socio-demographic characteristics of age and sex.

2.3. Measures

A self-completion questionnaire package was put together comprising a demographic cover sheet and a range of measures chosen in line with the variables under examination in the study (Appendix III). On average, these questionnaires took approximately 30-45 minutes to complete.

2.3.1. Demographic Cover Sheet

The demographic information collected consisted of gender, age, educational level, occupation and marital status. Participants were also asked about how long they had had chronic headache and what type of headache they experienced.

In addition, current and average pain intensity was measured using the Numerical Rating Scale-101 (NRS-101) recommended by Jensen, Karoly & Brave (1986). The NRS-101 is a 10 cm visual analogue scale (VAS). Participants were asked to indicate the level of their pain along the scale. The end points of the NRS-101 were anchored with the verbal descriptors 'no pain' and 'worst pain possible'. Jensen and colleagues reviewed a number of different rating scales and recommended the NRS-101 due to its simplicity, sensitivity and applicability across a wide age range.

2.3.2. Headache Disability Inventory (HDI)

The HDI (Jacobson, Ramadan, Aggarwal & Newman, 1994) is a 25-item self-assessment scale consisting of two subscales designed to measure the functional and emotional disabling effects of headache. Twelve items assess the effect of headaches on emotional functioning (e.g.: "My headaches make me angry", "I feel desperate because of my headaches", "I am afraid to go outside when I feel that a headache is starting"). Thirteen items assess the impact of headaches on daily activities (e.g. "I am unable to think clearly because of my headaches", "Because of my headaches I am less likely to socialise", "Because of my headaches I feel restricted in performing my routine daily activities"). Respondents are asked to answer each item with either a 'yes' (four points), 'sometimes' (two points), or 'no' (zero points) response. A maximum score of 100 points reflects severe self-perceived headache disability. The

HDI also includes two items that asses the frequency (1= '1 per month', 2= 'more than 1 but less than four per month', 3= 'more than 1 per week') and severity (1= 'mild', 2= 'moderate', 3= 'severe') of headaches.

The HDI has been shown to have high internal consistency and good content validity (Jacobson et al., 1994). The short-term (1 week) test-retest reliability of the emotional subscale (r = 0.95), functional subscale (r = 0.93) and total scale (r = 0.95) are robust. The long-term (2 months) test-retest stability of the emotional subscale (r = 0.82), functional subscale (r = 0.76) and total scale (r = 0.83) are also satisfactory. Patient reports on the HDI appear to be reasonably congruent with spouse reports (Jacobson, Ramadan, Norris & Newman, 1995). The HDI has been frequently used in headache research (e.g. French, Holroyd, Pinell, Malinoski, O'Donnell & Hill, 2000; Holroyd, Malinoski, Davis & Lipchik, 1999; Magnusson & Becker, 2003).

2.3.3. The Basic Emotions Scale (BES)

The Basic Emotions Scale (Power, 2003) is a self-report questionnaire designed to measure an individual's experience of a theoretically-derived list of emotions, consisting of five basic emotions (anger, sadness, disgust, fear and happiness) and additional related emotions. It comprises three parts, each containing 20 emotion terms which make up five subscales: anger (anger, frustration, irritation, aggression), sadness (despair, misery, gloominess, mournful), disgust (shame, guilt, humiliated, blameworthy), fear (anxiety, nervousness, tense, worried), and happiness (happiness, joy, loving, cheerful).

In Part 1 respondents are asked to rate on a seven-point scale (from 1 = never to 7 = very often) how often they have experienced each of the 20 emotions 'during the last week'. This provides a state-like judgement of emotion frequency over a recent timeframe. Similarly, in Part 2 respondents are asked to indicate how much 'in general' they experience those particular emotions. This provides a trait-like judgement of emotion frequency. In Part 3 respondents are asked to indicate 'how well they cope' with each emotion. The end points of the seven-point scale are "cope very well" and "cope very badly", with higher scores indicating difficulties to cope.

Internal reliability analysis for each of the 5 subscales showed good internal consistency, with Cronbach alpha values ranging from 0.790 to 0.842 (Power, 2003). The construct validity of the Basic Emotion Scale was assessed in a study whereby data collected through the Basic Emotions Scale was used to test six theoretical models of emotion (Power, 2003). Confirmatory factor analysis demonstrated that the inter-correlated basic emotions model (Power & Dalgleish, 1997) fitted the data, indicating that the Basic Emotions Scale is a valid self-report measure of emotion.

2.3.4. The Emotion Regulation Questionnaire (ERQ)

The Emotion Regulation Questionnaire is a self-report measure developed by Phillips (2003) based on a conceptual model of emotion regulation. In this model, emotional regulatory strategies were conceptualised as dysfunctional-versus-functional, which draw on internal-versus-external resources. The model postulates that dysfunctional strategies involve a rejection of unwanted emotional experiences, whereas functional strategies involve accepting these experiences.

The ERQ comprises two sections: section 1 is a 19-item measure designed to assess how individuals generally respond to their emotions. Respondents are asked to rate themselves on a five-point scale (from 0 = never to 4 = always, with negative items being scored in reverse order). This section of the ERQ assesses the frequency with which internal-dysfunctional (e.g. "I keep the feeling locked up inside"), internal-functional (e.g. "I put the situation into perspective"), external-dysfunctional (e.g. "I take my feelings out on others verbally") and external-functional (e.g. "I ask others for advice") emotion regulation strategies are used by respondents to regulate their emotions. Section 2 is a 24-item measure designed to assess the frequency with which respondents use the above mentioned emotion regulation strategies to regulate six particular emotions (sadness, happiness, shame, anger, anxiety, guilt).

The ERQ has been shown to have good internal consistency, with Cronbach alphas for the subscales ranging from 0.659 to 0.758. The convergent and construct validity of the ERQ were demonstrated in a study investigating quality of life in a population of normal adolescents (Phillips, 2003), where the scales of the ERQ were found to be significantly correlated with measures of quality of life and psychosomatic symptoms. Additionally, in the same study significant correlations were found between emotion regulation strategies and emotion-related seizure precipitants in a sample of adolescents with epilepsy.

2.3.5. State-Trait Anger Expression Inventory (STAXI)

The STAXI (Spielberger, Johnson, Russell, Crane, Jacobs, & Worden, 1985; Spielberger, 1988) is a 44-item measure, divided into three parts. Each item is rated on a four-point scale with the response options varying over the three parts.

The STAXI forms six scales and two subscales. The State Anger (S-Anger) scale measures the intensity of angry feelings at a particular time. The Trait Anger (T-Anger) scale measures individual differences in the disposition to experience anger. The Trait Anger scale can produce two subscales: Angry-Temperament (T-Anger/T), which measures the general propensity to experience anger without specific provocation; and Angry Reaction (T-Anger/R), which measures the general propensity to express anger when criticised or treated unfairly by others. The Anger-In (AX-In) scale measures the tendency to experience but suppress angry feelings. The Anger-Out (AX-Out) scale measures the tendency to express anger through aggressive behaviour towards other people and objects. The Anger Control (AX-Con) scale measures the tendency to attempt to control the experience and expression of angry feelings. The Anger Expression (AX/EX) scale comprises all the items of the AX-In, AX-Out and AX-Con scales, providing a general index of how often anger is expressed, regardless of the direction of the expression. The STAXI has good reliability and validity, with Cronbach alpha values for the scales ranging from 0.73 to 0.84, which shows satisfactory internal consistency (Spielberger et al., 1985).

The STAXI is frequently used in research to measure angry feelings in chronic pain patients, including headache sufferers (e.g. Asmundson, Wright, Norton & Veloso, 2001; Gaskin, Greene, Robinson & Geisser, 1992; Nicholson, Gramling, Ong, & Buenevar, 2003).

2.3.6. The Beck Depression Inventory – 2nd edition (BDI-II)

The BDI-II (Beck, Steer & Brown, 1996) is a 21-item self-report measure designed to assess the presence and severity of depressive symptoms in adults and adolescents. Each item comprises 4 representative statements related to depressive symptoms and

attitudes, reflecting a continuum of severity indicated by an ascending score from 0-3. The measure requires individuals to select the statement that best describes the way they have been feeling during the 'past two weeks'. A total score is derived by summing up all item scores. This summed score (range 0-63) indicates the severity of depressive symptoms. For example, scores of <13 indicate minimal levels of depression, scores of 14-19 indicate mild levels of depression, scores of 20-28 indicate moderate levels of depression, and scores of 29-63 indicate severe levels of depression. The BDI-II has good internal consistency, convergent validity and test-retest reliability (Beck, Steer & Brown, 1996), and it has been used extensively in headache research (e.g. Magnusson & Becker, 2003; Holroyd, Malinoski, Davis & Lipchik, 1999).

2.3.7. The Beck Anxiety Inventory (BAI)

The Beck Anxiety Inventory (Beck & Steer, 1993) is a 21-item self-report measure designed to assess the severity of anxiety symptoms. Each BAI item is rated on a four-point scale (from 0 = 'not at all' to 3 = 'severely, I could barely stand it'). The summed score on all items of the BAI (range 0-63) indicate the severity of anxiety symptoms. Scores of <7 indicate minimal levels of anxiety, scores of 8-15 indicate mild levels of anxiety, scores of 16-25 indicate moderate levels of anxiety, and scores of 26-63 indicate severe levels of anxiety. The Beck Anxiety Inventory has good internal consistency, convergent validity and stability (Beck & Steer, 1993), and is commonly used to measure anxiety in chronic pain populations (e.g. Hadjistavropoulos & La Chapelle, 2000; Kermit, Devine & Tatman, 2000).

2.4. Procedure

Data were collected within the period between January and June 2004 to fit in with the timescale requirements for the completion of the study. Patients attending the Headache Clinic at the Western General Hospital for a scheduled appointment were approached initially by the clinic specialist. Those referred from the Headache Clinic to the clinical psychology department for an initial assessment interview were first approached by the psychologist carrying out the assessment. At this stage, 2 patients did not meet the inclusion criteria and were not asked to take part in the investigation. Participants were first provided with verbal and written explanations of the research and its purpose. At this stage, 3 patients refused to take part in the study. Written informed consent (Appendix II) to be involved in the study was obtained from each patient who agreed to participate. Following this, the principal researcher gave participants a questionnaire package with the option to complete it there and then, or take it away to be returned by post in the stamped addressed envelope provided. Those who were able to spare the time were accompanied to a quiet area, where they could complete the questionnaires. The principal researcher remained available to answer any questions. Due to time constraints 37 patients opted to take the questionnaires away to be returned by post. Out of these, a total of 17 questionnaires were returned.

Control participants were approached directly by the principal researcher and other collaborators including friends and the clinical supervisor. Individuals approached were first asked about their general health status, in order to make sure they did not have problems with headaches. Those who met the criteria for inclusion in the study, were first given verbal and written explanations of the investigation. They were then

given the questionnaire package and instructed to return the complete questionnaires, and signed consent form, in the stamped addressed envelope provided. Of those who agreed to take part in the study, 16 did not return their questionnaires.

2.5. Data handling and analysis

The Statistical Package for the Social Sciences (SPSS) version 12.0 was used for all analysis. Comparative analysis was used to examine individual differences between headache and non-headache groups. Correlational and multiple regression analyses were used to explore the relationship between variables. Power was set at 0.8 and alpha at 0.05. Based on Clark-Carter's Power Tables (Clark-Carter, 1997), for a between subjects design with a power of 0.8 and an expected effect size of 0.6, the sample required was of 35 participants in each group.

A missing variable analysis (MVA) was carried out to determine the percentage of missing values in each variable (Appendix IV). With a few exceptions, the percentage of missing values was very small (< 4%) and these were randomly distributed. For the BDI-II and BAI, the procedure adopted to deal with missing values was the one recommended in the respective manuals, where questionnaires with up to 4 out of 21 items missing were entered in the analysis. For the other variables, the SPSS default option was used, whereby missing data were not included. Throughout the Results section, specific cases with a higher percentage of missing data will be pointed out.

Prior to data analysis all variables were screened for normality in line with the requirements for the use of parametric tests. Taking into consideration that the

groups were selected on the basis of being distinct, it was anticipated that some variables (e.g. headache-related variables, BDI-II and BAI) would depart from normality. Departures from normality in these variables were meaningful, therefore these data were not submitted to transformation, as this would hinder interpretation (Tabachinick & Fidell, 2001). For most of the other variables, skewness or kurtosis values fell within acceptable limits, i.e., less than twice its standard error. It was noted that the variables that presented problems of skewness and kurtosis, were subscales on a scale where most of the other variables had a satisfactory distribution. As such, these variables were not submitted to transformations with the purpose of maintaining consistency within the respective scale, bearing in mind the robustness of parametric tests, which are known to be quite accurate even when some of their assumptions are violated (Clark-Carter, 2004). Frequency tables and complete correlation matrices are contained in Appendix IV.

It is usual to apply Bonferroni corrections when doing multiple statistical tests. However, because the hypotheses in this study were directional this procedure was not used to decrease the risk of committing Type II errors (Clark-Carter, 2004; Howell, 1987).

3. Results

A total of 104 individuals met the criteria for inclusion in this study. The headache group comprised 57 participants (54.8%) and the control group comprised 47 participants (45.2 %). Tables 3.1 and 3.2 summarise the socio-demographic characteristics of both groups, as well as of the overall sample. Tables 3.3 to 3.7 summarise the clinical characteristics of the two groups.

3.1. Socio-demographic characteristics

Several individuals did not complete some of the demographic fields, as indicated where appropriate. Data regarding educational level in particular was the most affected with 36 individuals (34.6 %) failing to provide such information.

3.1.1. Age and Sex

The mean age of the total sample was 41.27 (SD = 13.64), with a minimum age of 20 years, and a maximum of 71 years. The majority of the participants were female. Taking into consideration that the control group was age- and sex-matched to the headache group, the higher percentage of females to males in this sample reflects the usual ratio of female to male headache sufferers documented in the literature (e.g. Lipton & Stewart, 1993; Schwartz, Stewart, & Lipton, 1998). Table 3.1. summarises the age and sex characteristics per group and overall sample. Analysis of potential group differences on these demographic variables indicated that no significant differences existed between the two groups in terms of either age ($t = 0.948$, $df = 102$, $p = 0.346$) or sex ($\chi^2 = 0.358$, $df = 1$, $p = 0.550$).

Table 3.1. Age and Sex per group and overall sample.

Demographics	Headache (n = 57)	Control (n = 47)	Overall (n = 104)
Age			
Mean	42.42	39.87	41.27
SD	14.08	13.11	13.64
Sex			
Male (%)	14 (24.56 %)	14 (29.79%)	28 (26.92%)
Female (%)	43 (75.43 %)	33 (70.21%)	76 (73.08%)

3.1.2. Marital Status, Educational Level and Occupational status

Most of the participants were married or co-habiting, and were currently employed. The majority was found to be educated at university level, although this information may not be accurate since a reasonable percentage of participants did not complete this field in the demographic cover sheet. Table 3.2 summarises these data for the whole sample and the two groups. Analysis of potential group differences on these demographic variables demonstrated that there were no significant differences in marital status ($\chi^2 = 2.281$, df = 3, p = 0.516), level of education ($\chi^2 = 4.529$, df = 2, p = 0.104), or occupational status ($\chi^2 = 3.153$, df = 4, p = 0.533).

Table 3.2. Marital Status, Educational Level and Occupational Status per group and overall sample.

Demographics	Headache N (%)	Control N (%)	Overall N (%)
Marital Status			
Single	17 (29.8%)	18 (38.3%)	35 (33.7%)
Married/Co-habiting	31 (54.4%)	23 (48.9%)	54 (51.9%)
Divorced	5 (8.8%)	2 (4.3%)	7 (6.7%)
Widowed	1 (1.8%)	_	1 (1.0%)
Missing	3 (5.3%)	4 (8.5%)	7 (6.7%)
Total	57 (100%)	47 (100%)	104 (100%)
Education			
School	11 (19.3%)	5 (10.6%)	16 (15.4%)
College/Vocational	3 (5.3%)	5 (10.6%)	8 (7.7%)
University	17 (29.8%)	27 (57.4%)	44 (64.75%)
Missing	26 (45.6%)	10 (21.3%)	36 (34.6%)
Total	57 (100%)	47 (100%)	104 (100%)
Occupation			
Unemployed	6 (10.5%)	1 (2.1%)	7 (6.7%)
Student	5 (8.8%)	6 (12.8%)	11 (10.6%)
Employed	31 (54.4%)	26 (55.3%)	57 (54.8%)
Housewife	3 (5.3%)	2 (4.3%)	5 (4.8%)
Retired	9 (15.8%)	8 (17.0%)	17 (16.3%)
Missing	3 (5.3%)	4 (8.5%)	7 (6.7%)
Total	57 (100%)	47 (100%)	104 (100%)

3.2. Clinical characteristics

The inclusion and exclusion criteria applied at the recruitment stage, meant that the two groups were expected to be significantly different in their clinical characteristics. In order to test this assumption, independent samples t-tests and chi-square tests were carried out to compare the scores of the two groups in the variables described below.

3.2.1. Years of suffering from headaches

As summarised in Table 3.3, none of the control participants suffered from recurrent headaches. In contrast, the headache participants had been suffering from headaches for 16.36 years on average, therefore the difference between the groups was found to be highly significant ($t = 8.265$, $df = 98$, $p < 0.001$).

Table 3.3. Years of headaches

Clinical Characteristic	Groups	N	Missing	Min	Max	Mean	SD
Years of headaches	Headache	53	4	0.11	50.90	16.36	13.56
	Control	47	0	0.00	0.00	0.00	0.00

3.2.2. Frequency of headache

The frequency with which participants experienced headache was measured by the statement "I have headache", which accompanies the HDI. In this item, respondents choose between 3 categories: "1 per month', 'more than 1 but less than 4 per month', and 'more than 1 per week'. Since headache is a common ailment, it was expected that control participants would have some experience of headache, though not of a chronic nature. Control participants who experienced headaches less often than the given choices, were instructed to write "no recurrent headache" beside this item. Table 3.4 shows the results for the two groups. As expected, the Headache group reported experiencing headaches significantly more often than the Control group ($\chi^2 = 79.296$; $df = 3$, $p < 0.001$).

Table 3.4. Frequency of headache experience for Headache (N=54) and Control (N=47) groups.

"I have headache"	Groups	Frequency	%
"No recurrent headache"	Headache	_	_
	Control	31	66.0
"1 per month"	Headache	3	5.6
	Control	12	25.5
"> 1 but < 4 per month"	Headache	12	22.2
	Control	4	8.5
"More than 1 per week"	Headache	39	72.2
	Control	_	_

3.2.3. Severity of headache

Three measures of headache severity were initially included in this study. One of these measures was the item "My headache is..." that goes with the HDI. The respondents were asked to choose between three categories: "mild", "moderate" and "severe". As with the frequency item, control participants who experienced lower levels of headache were instructed to write "no recurrent headache" beside this item. Table 3.5 summarises the results. The majority of the headache sufferers in this sample reported experiencing severe headaches. As expected, the two groups differed significantly in terms of headache intensity ($\chi^2 = 83.794$, df = 3, $p < 0.001$).

Table 3.5. Severity of headache experience for Headache (N=52) and Control (N=46) groups.

"My headache is"	Groups	Frequency	%
"No recurrent headache"	Headache	_	_
	Control	25	54.3
"Mild"	Headache	1	1.9
	Control	18	39.1
"Moderate"	Headache	19	36.5
	Control	3	6.5
"Severe"	Headache	32	61.5
	Control	_	_

The other two measures of headache severity were the NRS-101 Visual Analogue Scales (VAS) items, included in the demographic cover sheet. Participants were asked to indicate on a 10 cm line the level of their pain "currently" and "on average". The end points were "no pain" and " worst pain possible". Results are summarised in Table 3.6. As expected, a significant difference was found between the groups in terms of pain levels "currently" ($t = 8.680$, $df = 56.731$, $p < 0.001$) and "on average" ($t = 20.920$, $df = 68.181$, $p < 0.001$).

Assessing current pain state is particularly relevant as it has been demonstrated that high pain levels, at the time of completing questionnaires, tend to increase scores on measures of psychological symptoms (e.g. Holroyd, France, Nash & Hursey, 1993). A paired samples t-test indicated that the level of pain the headache participants were experiencing, at the time of completing the questionnaires, was significantly lower than their average pain levels ($t = 5.505$, $df = 56$, $p < 0.001$). This suggests that current pain state might not have been an artefact in the study.

In order to ensure that multicollinearity did not exist, the HDI measure of severity "My headache is..." was dropped from subsequent multiple regression analyses in favour of the NRS-101 (VAS) that measured pain levels "on average". This seemed appropriate considering that the former had 5.8% missing values while the later had none. Moreover, the HDI item was highly correlated with the NRS-101 ($r = 0.841$, $n = 98$, $p < 0.01$) indicating that they contained redundant information and were not needed in the same analysis (Tabachnick & Fidell, 2001).

Table 3.6. "Current" and "on average" pain level per group

NRS-101 (VAS)	Groups	N	Missing	Min	Max	Mean	SD
Current pain level	Headache	57	0	0	10	3.95	3.37
	Control	47	0	0	1	0.64	0.25
Average pain level	Headache	57	0	1	10	6.77	2.24
	Control	47	0	0	3	0.19	0.68

NRS-101 (VAS) = Visual Analogue Scale

3.2.4. Type of headache

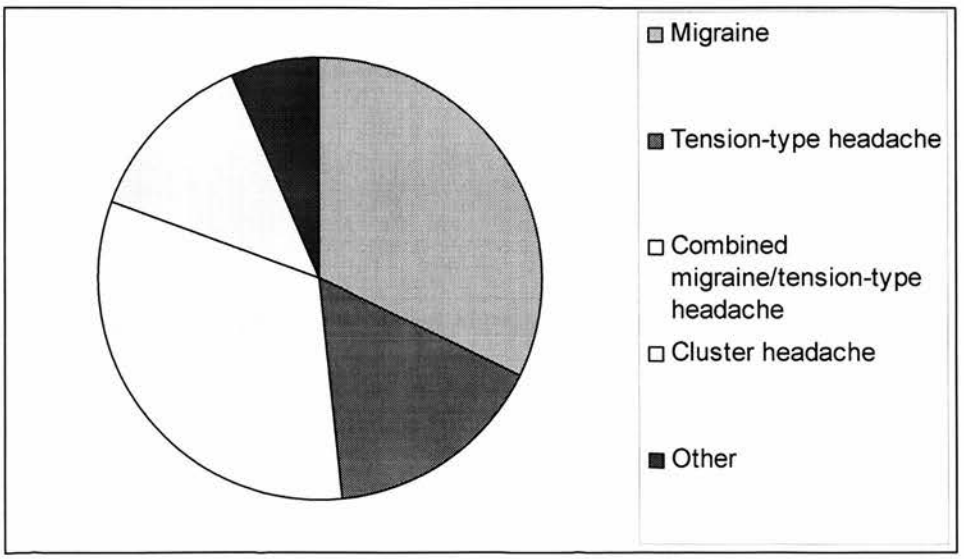
The most frequent types of headache disorders were *migraine* and *combined migraine/tension-type headache*. Five individuals reported experiencing more than one type of headache. For instance, *cluster headache* with *combined migraine/tension-type headache* (n=2), *cluster headache* with *migraine* (n=1), *cluster headache* with *tension-type headache* (n=1), and *combined migraine/tension-type headache* with "other" type of headache (n=1, in this case "sinus headache"). Three individuals reported suffering from "other" types of headache: "cloudy head" (n=1), "daily headache" (n=1) and "not diagnosed" (n=1). None of the control participants reported suffering from any type of primary headache disorder. Table 3.7

summarises the percentage of participants who gave a "yes" response to that item. Figure 3.1 shows the percentage of individuals in the headache group who reported suffering from each headache disorder.

Table 3.7. Distribution of type of headache disorder per group.

Headache disorder	Groups	N	% yes
Migraine	Headache	20	35.1
Tension-Type Headache	Headache	10	17.5
	Control	_	_
Combined migraine/tension-type headache	Headache	20	35.1
	Control	_	_
Cluster headache	Headache	8	14
	Control	_	_
Other	Headache	4	14.0
	Control	_	_

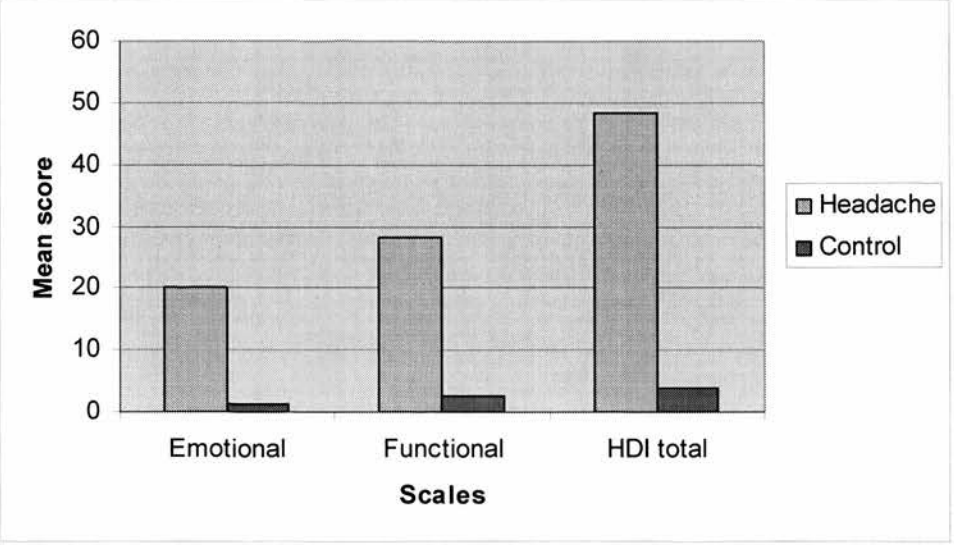
Figure 3.1. Distribution of headache disorders within the headache group.



3.3. Headache-related disability

The Headache Disability Inventory (HDI) assesses the self-perceived emotional and functional impact of chronic headache disorders. As shown in Figure 3.2, the functional disabling effect of headaches was reported to be higher in comparison with the emotional. As shown in Table 3.8, independent samples t-tests demonstrated that the two groups differed significantly in terms of headache-related disability.

Figure 3.2. Mean scores on the Headache Disability Inventory (HDI)



HDI = Headache Disability Inventory.

Table 3.8. Comparison between groups on the Headache Disability Inventory (HDI).

HDI	Group	N	Mean	SD	t value	Degrees of freedom	Significance (2-tailed)
Emotional	Headache	57	20.14	11.05	12.380	65.204	$p < 0.001$
	Control	47	1.28	2.90			
Functional	Headache	57	28.28	10.15	16.378	88.599	$p < 0.001$
	Control	47	2.72	5.43			
HDI total	Headache	57	48.42	19.42	15.793	76.502	$p < 0.001$
	Control	47	4.00	7.80			

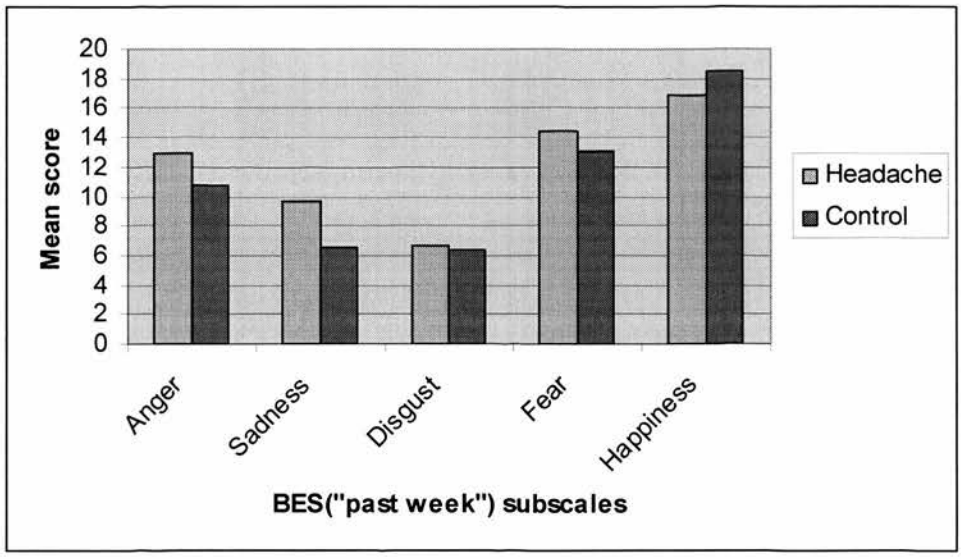
3.4. Comparing the experience of basic emotions between the groups

The Basic Emotions Scale (BES) was used to assess the frequency with which the participants experienced the basic emotions of anger, sadness, disgust, fear and happiness. Additionally, it also measured how they usually cope with these emotions. Independent samples t-tests were carried out to compare the mean scores of each group. Degrees of freedom that are less than a whole number, indicate that the Levene's test for equality of variances revealed unequal variances between the two means. Two-tailed significance values were halved when testing predicted variables, namely anger, sadness and fear.

3.4.1. BES – Part 1: Experience of basic emotions "in the past week".

This measure provides a state-like judgement of emotion frequency over a recent period of time. As shown in Figure 3.3, the basic emotions of happiness, fear and anger were reported as the most frequently experienced by both groups. The headache group scored higher on all negative emotions scales, and lower on the positive emotions scale. As summarised in Table 3.9, the headache group scored significantly higher on the anger ($t = 2.280$, $df = 100.908$, $p < 0.05$, one-tailed) and sadness ($t = 4.022$, $df = 89.675$, $p < 0.001$, one-tailed) scales.

Figure 3.3. Basic emotions experienced "in the past week".



BES = Basic Emotions Scale.

Table 3.9. Comparison between groups on the BES-Part 1.

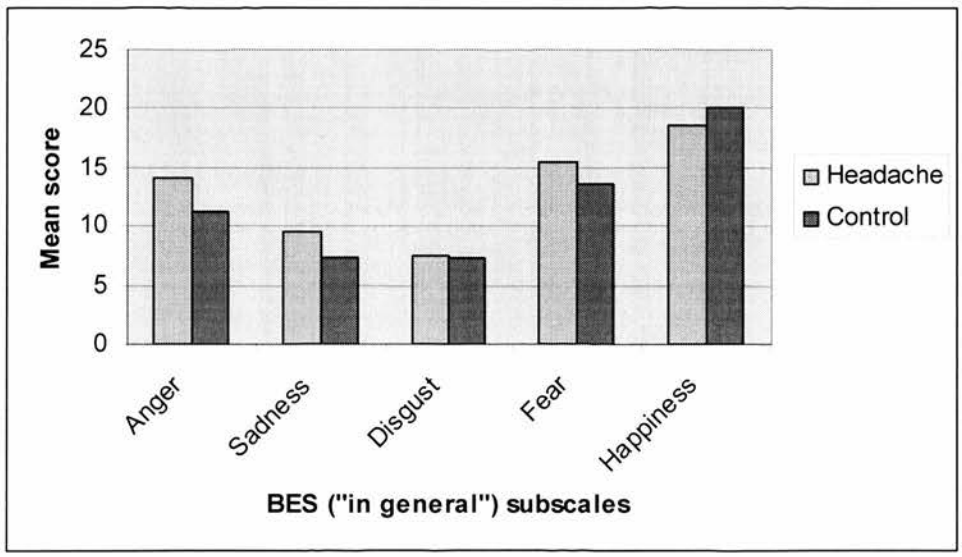
BES ("in the past week")	Group	N	Mean	SD	t value	Degrees of freedom	Significance
Anger	Headache	57	12.86	5.48	2.280	100.908	$p < 0.05$
	Control	47	10.72	4.06			
Sadness	Headache	57	9.63	5.05	4.022	89.675	$p < 0.001$
	Control	47	6.49	2.76			
Disgust	Headache	57	6.63	3.85	0.390	102	$p = 0.697$
	Control	47	6.36	3.05			
Fear	Headache	57	14.42	5.72	1.340	102	$p = 0.09$
	Control	47	13.04	4.53			
Happiness	Headache	57	16.86	5.44	1.679	102	$p = 0.096$
	Control	47	18.55	4.70			

BES = Basic Emotions Scale.

3.4.2. BES – Part 2: Experience of basic emotions "in general".

This measure provides a trait-like judgement of emotion frequency. As shown in Figure 3.4, the basic emotions of happiness, fear and anger were reported as the most frequently experienced in general by both groups. The headache group scored higher on all negative emotions scales, and lower on the positive emotions scale. As summarised in Table 3.10, the headache group scored significantly higher on the anger ($t = 3.488$, $df = 100.276$, $p < 0.001$, one-tailed), sadness ($t = 2.591$, $df = 100.015$, $p < 0.001$, one-tailed) and fear ($t = 1.902$, $df = 102$, $p < 0.05$, one-tailed) scales.

Figure 3.4. Basic emotions experienced "in general".



BES = Basic Emotions Scale.

Table 3.10. Comparison between groups on the BES-Part 2.

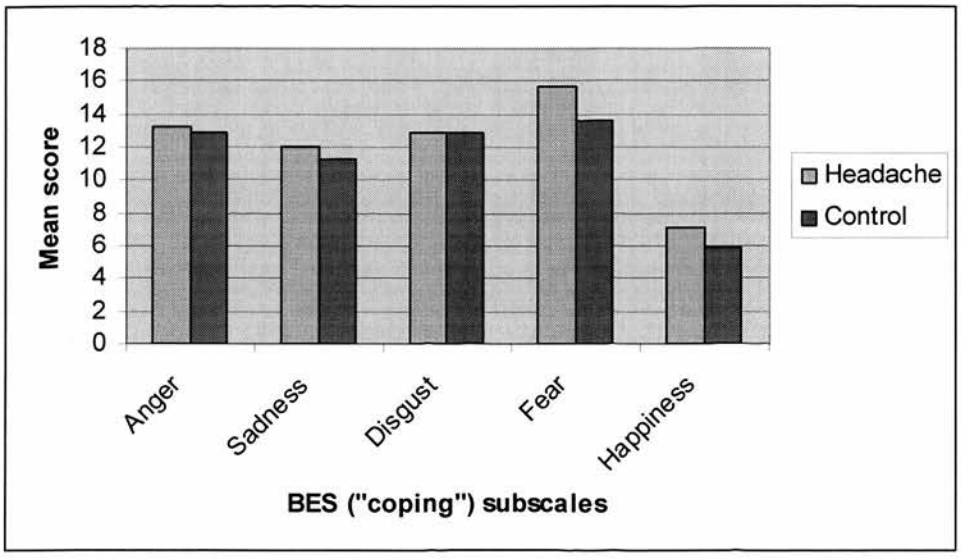
BES ("in general")	Group	N	Mean	SD	t value	Degrees of freedom	Significance
Anger	Headache	57	14.05	4.92	3.488	100.276	$p < 0.001$
	Control	47	11.15	3.54			
Sadness	Headache	57	9.49	4.83	2.591	100.015	$p < 0.001$
	Control	47	7.38	3.44			
Disgust	Headache	57	7.42	4.41	0.226	102	$p = 0.822$
	Control	47	7.23	3.93			
Fear	Headache	57	15.40	5.09	1.902	102	$p < 0.05$
	Control	47	13.66	4.06			
Happiness	Headache	57	18.50	5.63	1.546	102	$p = 0.125$
	Control	47	20.15	5.07			

BES = Basic Emotions Scale.

3.4.3. BES – Part 3: Ability to cope with basic emotions.

In this section of the BES respondents indicated how well they coped when they experienced those emotions. The end points of the seven-point scale were "cope very well" and "cope very badly", with higher scores indicating a self-perceived inability to cope. Due to a photocopying mistake a total of eleven participants did not receive this part of the BES, therefore results presented are for a reduced sample (47 in the headache group and 46 in the control group). As shown in Figure 3.5 and Table 3.11, the headache group reported feeling less able to cope with virtually all emotions including happiness. Significant differences, however, were noted only for the emotion of fear ($t = 1.872$, $df = 80.291$, $p < 0.05$, one-tailed).

Figure 3.5. Inability to cope with basic emotions



BES = Basic Emotions Scale.

Table 3.11. Comparison between groups on the BES-Part 3.

BES (coping)	Group	N	Mean	SD	t value	Degrees of freedom	Significance
Anger	Headache	47	13.17	4.93	0.323	91	$p = 0.748$
	Control	46	12.85	4.70			
Sadness	Headache	47	11.96	5.70	0.692	91	$p = 0.491$
	Control	46	11.22	4.53			
Disgust	Headache	47	12.81	6.76	- 0.031	91	$p = 0.975$
	Control	46	12.85	5.27			
Fear	Headache	47	15.62	6.17	1.872	80.291	$p < 0.05$
	Control	46	13.59	4.11			
Happiness	Headache	47	7.13	3.75	1.827	83.661	$p = 0.071$
	Control	46	5.89	2.70			

BES = Basic Emotions Scale.

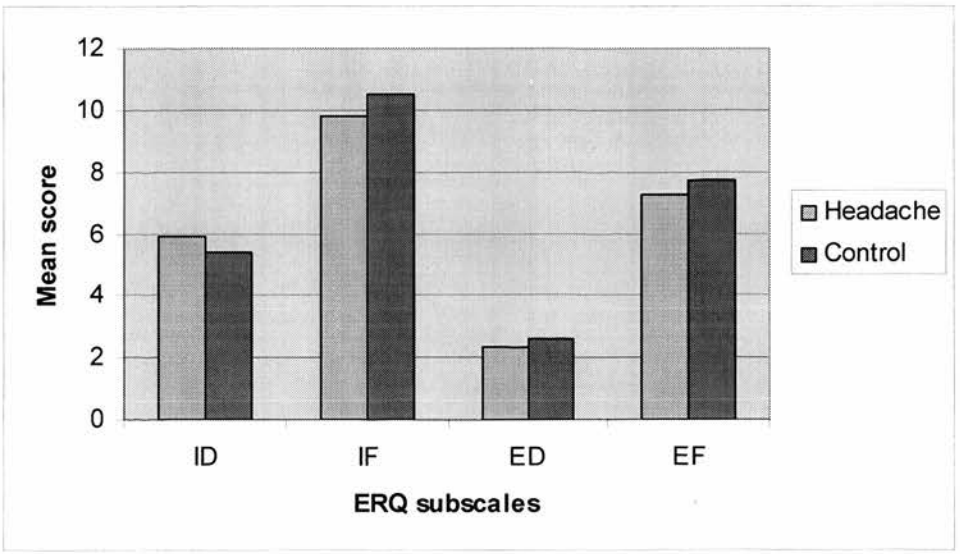
3.5. Comparing the strategies used to regulate emotions between groups

Independent samples t-tests were carried out to compare the mean scores of each group on the Emotion Regulation Questionnaire (ERQ) subscales: internal-dysfunctional (ID), internal-functional (IF), external-dysfunctional (ED) and external-functional (EF).

3.5.1. ERQ- Part 1: General response to emotions.

As shown in Figure 3.6, both groups reported using internal- and external-functional strategies more often than internal- and external-dysfunctional. The headache group reported making more use of internal-dysfunctional strategies to regulate emotions than the control group, but less use of external-dysfunctional strategies. In terms of the use of internal- and external-functional strategies, the headache sufferers scored lower than controls. However, none of these differences reached significance levels (see Table 3.12).

Figure 3.6. Strategies used to regulate emotions in general



ERQ = Emotion Regulation Questionnaire.
ID = internal-dysfunctional, IF = internal-functional, ED = external-dysfunctional, EF = external-functional.

Table 3.12. Comparison between groups on the ERQ-Part 1.

ERQ (Part 1)	Group	N	Mean	SD	t value	Degrees of freedom	Significance (2-tailed)
Internal-Dysfunctional	Headache	57	5.91	3.21	0.877	102	p = 0.383
	Control	47	5.38	2.88			
Internal-Functional	Headache	57	9.81	2.77	1.340	102	p = 0.183
	Control	47	10.53	2.72			
External-Dysfunctional	Headache	57	2.35	1.89	0.634	102	p = 0.528
	Control	47	2.60	2.04			
External-Functional	Headache	57	7.28	3.09	0.828	102	p = 0.409
	Control	47	7.74	2.51			

3.5.2. ERQ- Part 2: General response to particular emotions.

This part of the ERQ measures the frequency with which respondents use the above mentioned emotion regulation strategies to regulate six specific emotions (sadness, happiness, shame, anger, anxiety, guilt). The items corresponding to each strategy are described below.

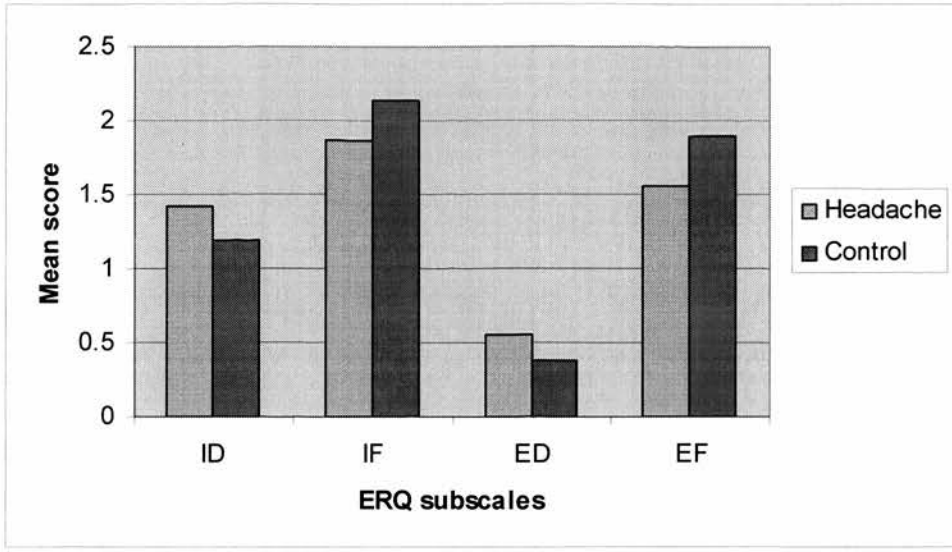
- Internal-dysfunctional: "I try not to let myself feel or express this."
- External-functional: "I seek social support."
- Internal-functional: "I change the way I view the situation."
- External-dysfunctional: "I take my feelings out on the people or objects around me."

3.5.2.1. Sadness

As shown in Figure 3.7, the headache group reported employing internal- and external-dysfunctional strategies to regulate the emotion sadness more often than did

the control group. Headache sufferers also reported using internal- and external-functional strategies less often than controls. However, none of these differences reached significance levels (see Table 3.13).

Figure 3.7. Distribution of strategies used to regulate Sadness



ERQ = Emotion Regulation Questionnaire
ID = internal-dysfunctional, IF = internal-functional, ED = external-dysfunctional, EF = external-functional.

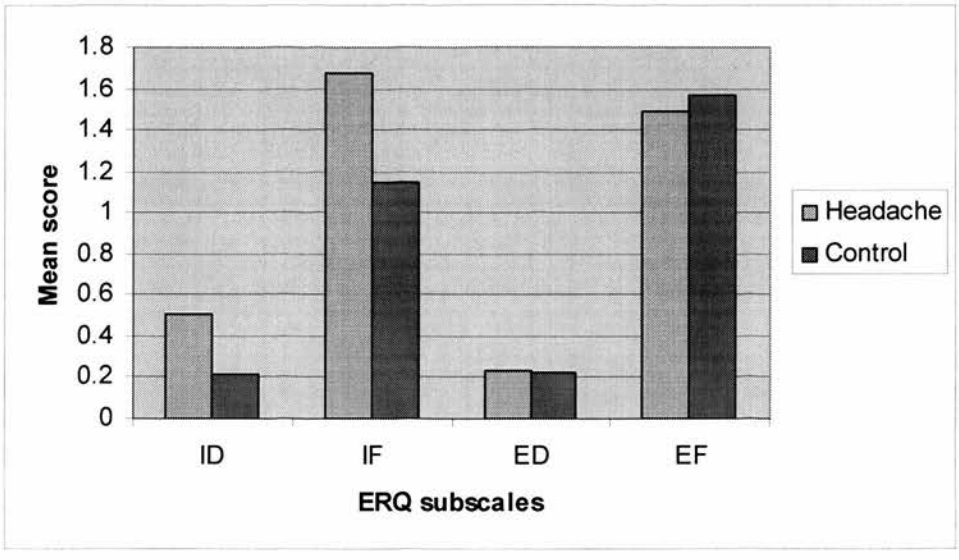
Table 3.13. Comparison between groups on strategies to regulate Sadness.

ERQ (Sadness)	Group	N	Mean	SD	t value	Degrees of freedom	Significance (2-tailed)
Internal-Dysfunctional	Headache	57	1.42	1.03	1.226	101.918	p = 0.223
	Control	47	1.19	0.88			
Internal-Functional	Headache	57	1.86	0.91	1.480	102	p = 0.142
	Control	47	2.13	0.92			
External-Dysfunctional	Headache	57	0.56	0.91	1.151	98.287	p = 0.235
	Control	47	0.38	0.61			
External-Functional	Headache	57	1.56	1.12	1.551	102	p = 0.124
	Control	47	1.89	1.05			

3.5.2.2. Happiness

As shown in Figure 3.8, the headache group reported employing internal-dysfunctional strategies to regulate the emotion happiness more often than did the control group, though not significantly. They also reported using external-dysfunctional strategies slightly more often. Headache sufferers reported using internal-functional strategies to regulate happiness significantly more often than controls ($t = 2.322$, $df = 102$, $p < 0.05$, two-tailed), whereas control participants reported using external-functional strategies more often, though not significantly (see Table 3.14).

Figure 3.8. Distribution of strategies used to regulate Happiness



ERQ = Emotion Regulation Questionnaire
ID = internal-dysfunctional, IF = internal-functional, ED = external-dysfunctional, EF = external-functional.

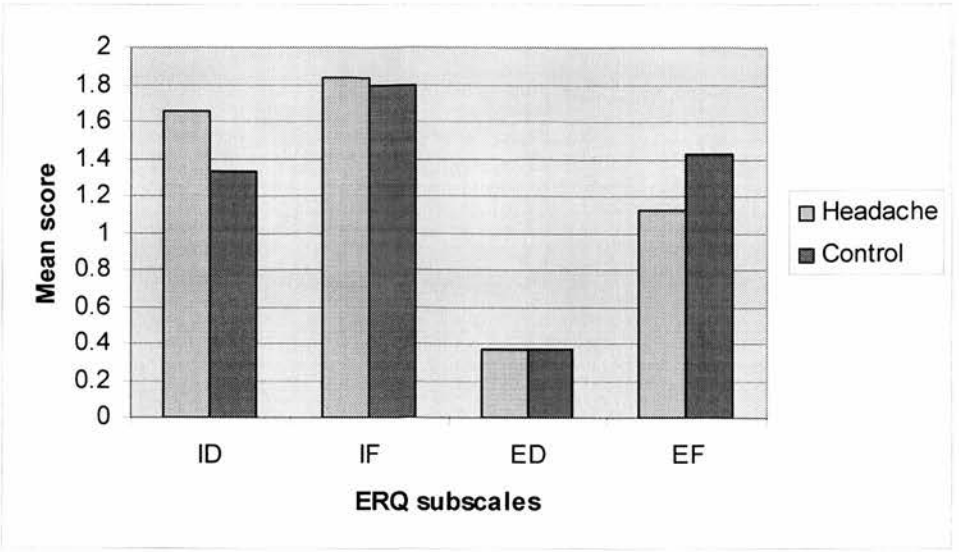
Table 3.14. Comparison between groups on strategies to regulate Happiness.

ERQ (Happiness)	Group	N	Mean	SD	t value	Degrees of freedom	Significance (2-tailed)
Internal-Dysfunctional	Headache	57	0.51	0.98	1.935	90.555	$p = 0.069$
	Control	47	0.21	0.55			
Internal-Functional	Headache	57	1.67	1.09	2.322	102	$p < 0.05$
	Control	47	1.15	1.18			
External-Dysfunctional	Headache	57	0.23	0.57	0.088	101	$p = 0.930$
	Control	46	0.22	0.66			
External-Functional	Headache	57	1.49	1.09	0.344	88.550	$p = 0.731$
	Control	47	1.57	1.33			

3.5.2.3. Shame

As shown in Figure 3.9, the headache group reported employing internal-dysfunctional strategies to regulate the emotion shame more often than did the control group. Headache sufferers also reported using internal-functional strategies slightly more often than controls, although controls made more use of external-functional strategies. However, none of these differences reached significance levels (see Table 3.15). Regarding the use of external-dysfunctional strategies, both groups scored equally in that subscale.

Figure 3.9. Distribution of strategies used to regulate Shame



ERQ = Emotion Regulation Questionnaire
ID = internal-dysfunctional, IF = internal-functional, ED = external-dysfunctional, EF = external-functional.

Table 3.15. Comparison between groups on strategies to regulate Shame.

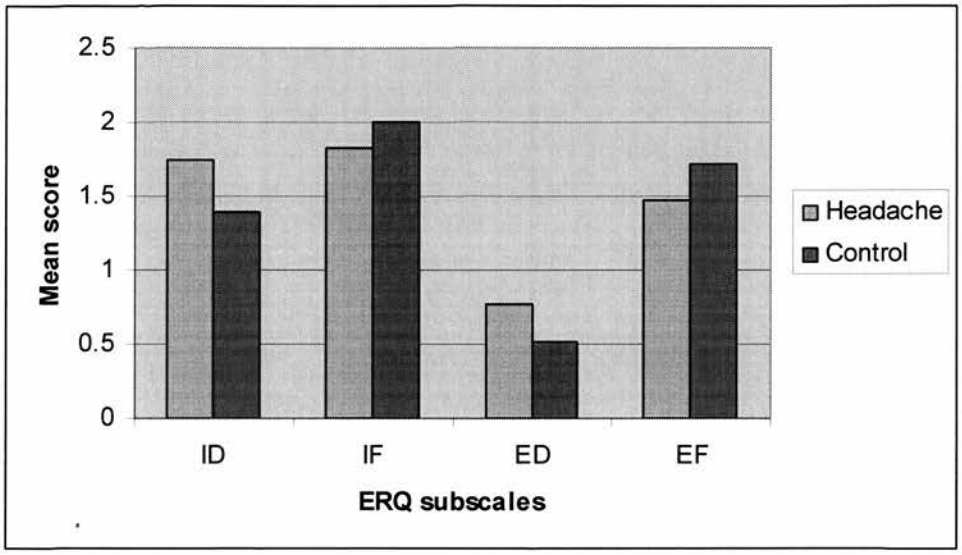
ERQ (Shame)	Group	N	Mean	SD	t value	Degrees of freedom	Significance (2-tailed)
Internal-Dysfunctional	Headache	57	1.65	1.27	1.523	98.498	p = 0.131
	Control	46	1.33	0.87			
Internal-Functional	Headache	57	1.84	0.88	0.218	101	p = 0.828
	Control	46	1.80	0.87			
External-Dysfunctional	Headache	57	0.37	0.67	0.009	101	p = 0.993
	Control	46	0.37	0.64			
External-Functional	Headache	57	1.12	0.93	1.636	101	p = 0.105
	Control	46	1.43	1.00			

3.5.2.4. Anger

As represented in Figure 3.10, headache sufferers reported using internal- and external-functional strategies to regulate anger less often than controls, though not significantly. As summarised in Table 3.16, the headache group reported employing

internal-dysfunctional strategies to regulate the emotion of anger significantly more often than did the control group ($t = 1.868$, $df = 101$, $p < 0.05$, one-tailed). They also reported using external-dysfunctional strategies more often, though not significantly.

Figure 3.10. Distribution of strategies used to regulate Anger



ERQ = Emotion Regulation Questionnaire
ID = internal-dysfunctional, IF = internal-functional, ED = external-dysfunctional, EF = external-functional.

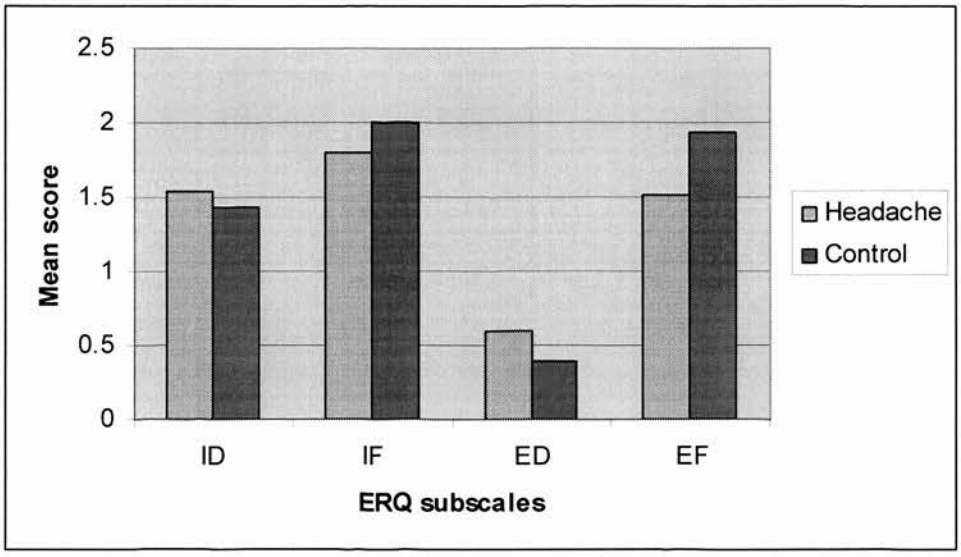
Table 3.16. Comparison between groups on strategies to regulate Anger.

ERQ (Anger)	Group	N	Mean	SD	t value	Degrees of freedom	Significance
Internal-Dysfunctional	Headache	57	1.74	0.97	1.868	101	$p < 0.05$
	Control	46	1.39	0.88			
Internal-Functional	Headache	57	1.82	0.83	1.110	101	$p = 0.270$
	Control	46	2.00	0.76			
External-Dysfunctional	Headache	57	0.77	0.98	1.542	97.911	$p = 0.126$
	Control	46	0.52	0.66			
External-Functional	Headache	57	1.47	1.02	1.192	101	$p = 0.236$
	Control	46	1.71	1.05			

3.5.2.5. Anxiety

As shown in Figure 3.11, the headache group reported employing internal- and external-dysfunctional strategies to regulate anxiety more often than did the control group, though not significantly. Inversely, the control group reported using internal-functional strategies more often than headache sufferers, though not significantly, and employing external-functional strategies significantly more often ($t = 2.028$, $df = 100$, $p < 0.05$, one-tailed), as shown in Table 3.17.

Figure 3.11. Distribution of strategies used to regulate Anxiety



ERQ = Emotion Regulation Questionnaire
ID = internal-dysfunctional, IF = internal-functional, ED = external-dysfunctional, EF = external-functional.

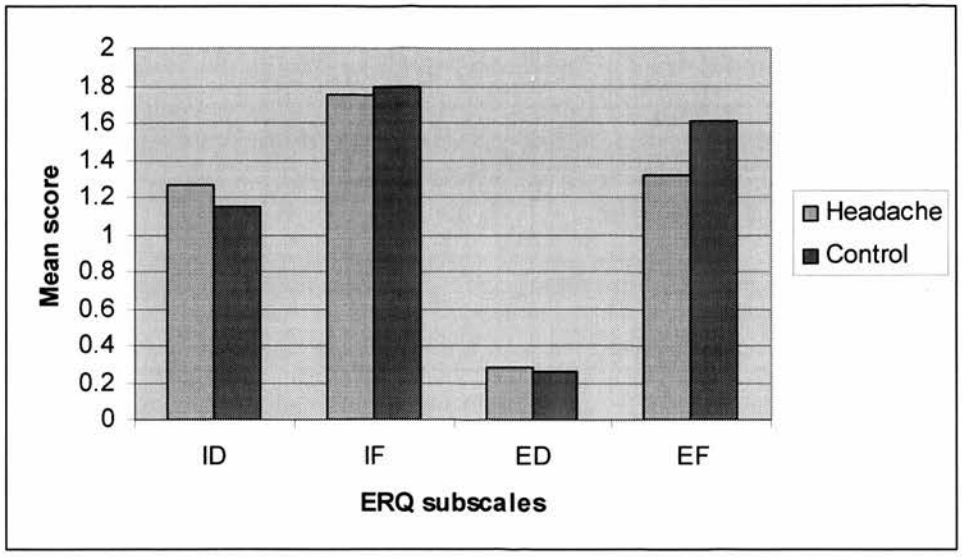
Table 3.17. Comparison between groups on strategies to regulate Anxiety.

ERQ (Anxiety)	Group	N	Mean	SD	t value	Degrees of freedom	Significance
Internal-Dysfunctional	Headache	57	1.54	0.93	0.572	101	$p = 0.568$
	Control	46	1.43	1.00			
Internal-Functional	Headache	57	1.80	0.83	1.179	101	$p = 0.241$
	Control	46	2.00	0.82			
External-Dysfunctional	Headache	57	0.60	1.00	1.220	101	$p = 0.203$
	Control	46	0.39	0.61			
External-Functional	Headache	56	1.52	1.03	2.028	100	$p < 0.05$
	Control	46	1.93	1.04			

3.5.2.6. Guilt

As represented in Figure 3.12, the headache group reported employing internal-dysfunctional strategies to regulate the emotion of guilt more often than did the control group. Their use of external-dysfunctional strategies was found to be only slightly higher than that of controls. Headache sufferers also reported using external-functional strategies less often than controls as well as slightly less internal-functional strategies. However, none of these differences reached significance levels (see Table 3.18).

Figure 3.12. Distribution of strategies used to regulate Guilt



ERQ = Emotion Regulation Questionnaire
ID = internal-dysfunctional, IF = internal-functional, ED = external-dysfunctional, EF = external-functional.

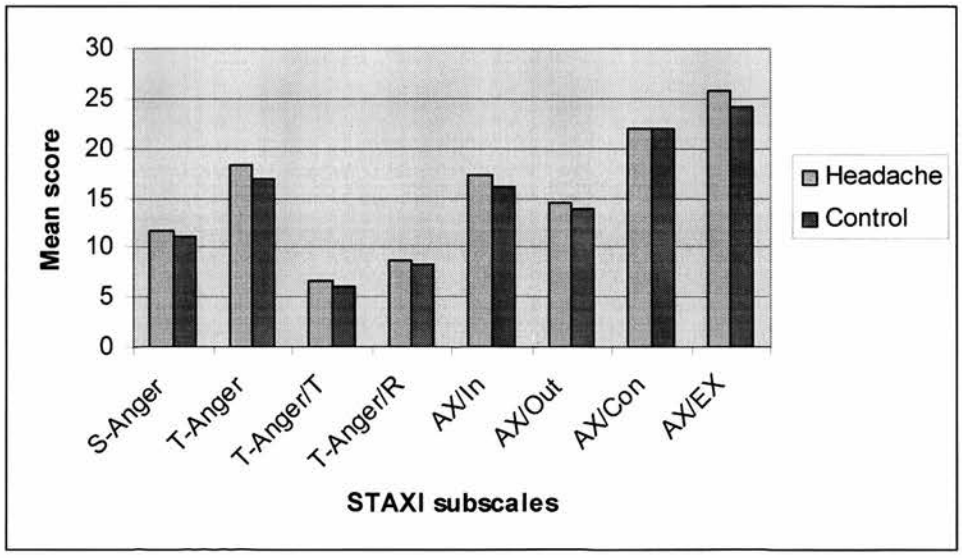
Table 3.18. Comparison between groups on strategies to regulate Guilt.

ERQ (Guilt)	Group	N	Mean	SD	t value	Degrees of freedom	Significance (2-tailed)
Internal-Dysfunctional	Headache	57	1.26	1.04	0.590	101	p = 0.557
	Control	46	1.15	0.82			
Internal-Functional	Headache	57	1.75	0.91	0.262	101	p = 0.794
	Control	46	1.80	1.02			
External-Dysfunctional	Headache	57	0.28	0.53	0.189	101	p = 0.851
	Control	46	0.26	0.53			
External-Functional	Headache	56	1.32	1.05	1.334	100	p = 0.185
	Control	46	1.61	1.12			

3.6. Comparing scores on the State-Trait Anger Expression Inventory (STAXI) between groups.

Figure 3.13 shows that the headache group scored higher than the control group on all anger subscales, although only slightly higher on the Anger Control (AX/Con). However, independent samples t-tests revealed that none of the differences were significant (see Table 3.19).

Figure 3.13. Distribution of mean scores on the STAXI subscales



S-Anger = State Anger, T-Anger = Trait Anger, T-Anger/T = Trait Anger Temperament, T-Anger/R = Trait Anger Reactive, AX/In = Anger in, AX/Out = Anger out, AX/Con = Anger Control, AX/EX = Anger Expression.

Table 3.19. Comparison between groups on the STAXI subscales.

STAXI subscales	Group	N	Mean	SD	t value	Degrees of freedom	Significance (2-tailed)
S-Anger	Headache	56	11.73	3.41	1.236	93.378	p = 0.220
	Control	44	11.05	2.11			
T-Anger	Headache	56	18.29	4.63	1.509	98	p = 0.134
	Control	44	16.98	3.84			
T-Anger/T	Headache	56	6.63	2.62	1.449	97.921	p = 0.150
	Control	44	5.95	1.10			
T-Anger/R	Headache	56	8.68	2.18	0.896	98	p = 0.372
	Control	44	8.30	2.04			
AX/In	Headache	56	17.23	4.20	1.263	98	p = 0.210
	Control	44	16.16	4.24			
AX/Out	Headache	56	14.43	3.21	0.462	98	p = 0.359
	Control	44	13.84	3.11			
AX/Con	Headache	56	21.91	5.59	0.023	98	p = 0.982
	Control	44	21.89	4.93			
AX/EX	Headache	56	25.75	8.95	1.040	97.892	p = 0.1505
	Control	44	24.11	6.78			

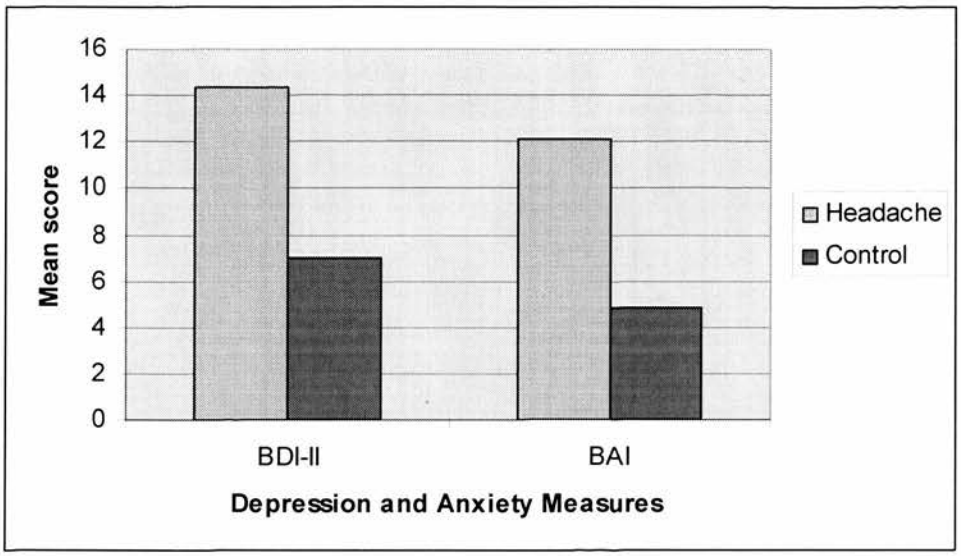
S-Anger = State Anger, T-Anger = Trait Anger, T-Anger/T = Trait Anger Temperament, T-Anger/R = Trait Anger Reactive, AX/In = Anger in, AX/Out = Anger out, AX/Con = Anger Control, AX/EX = Anger Expression.

3.7. Comparing levels of depression and anxiety between groups

Independent samples t-tests were carried out to compare the mean scores of each group on the Beck Depression Inventory (BDI-II) and the Beck Anxiety Inventory BAI). As represented in Figure 3.14 and summarised in Table 3.20, the headache group was found to have significantly higher levels of depression ($t = 4.879$, $df = 88.436$, $p < 0.001$, one-tailed) and anxiety ($t = 5.569$, $df = 84.722$, $p < 0.001$, one-tailed) than the control group. In both scales, the levels of anxiety and depression

were in the "mild" range for the headache group and in the "minimum" range for the control group.

Figure 3.14. Distribution of mean scores on the BDI-II and BAI



BDI-II = Beck Depression Inventory (2nd edition).
BAI = Beck Anxiety Inventory.

Table 3.20. Comparison between groups on the BDI-II and BAI.

Measures	Group	N	Mean	SD	t value	Degrees of freedom	Significance
BDI-II	Headache	55	14.40	9.50	4.879	88.436	$p < 0.001$
	Control	46	7.02	5.45			
BAI	Headache	56	12.13	8.53	5.569	84.722	$p < 0.001$
	Control	46	4.85	4.33			

BDI-II = Beck Depression Inventory (2nd edition).
BAI = Beck Anxiety Inventory.

3.8. Examining the relationship between variables

Pearson's test of correlation was used to examine the relationship between the psychological measures (BES, ERQ, STAXI, BAI, BDI-II) and the measures of

headache frequency, severity and disability. Although the Headache Disability Inventory (HDI) is organised into emotional and functional subscales, correlation analysis revealed that the total score (HDI total) was highly correlated with the emotional ($r = 0.960$, $n = 104$, $p < 0.01$) and functional ($r = 0.973$, $n = 104$, $p < 0.01$) subscales. This indicates that the total HDI score is singular with its subscales, therefore only the total score was included in the analysis, a procedure that has been used in previous studies (e.g. French, Holroyd, Pinell, Malinoski, O'Donnell & Hill, 2000; Holroyd, Malinoski, Davis & Lipchik, 1999).

3.8.1 Basic Emotions Scale (BES)

3.8.1.1. BES – Part 1 ("in the past week")

Table 3.21 displays the significant correlations found between the BES – Part 1 ("in the past week") and headache frequency, headache severity and HDI total for the headache group and the control group. In the headache group, the HDI total score was found to be positively correlated with the experience of anger, sadness, disgust and fear "in the past week". A negative correlation was found between the HDI total score and the emotion of happiness. A positive correlation was found between the severity of headache and sadness. The frequency of headache was positively correlated with the emotion of happiness. No significant correlations were found in the control group.

Table 3.21. Significant correlations between the BES – Part 1 ("in the past week") and headache frequency, headache severity and HDI total.

Measures by group	BES – Part 1 ("In the past week")				
	Anger	**Sadness**	**Disgust**	**Fear**	**Happiness**
Headache					
Headache frequency	–	–	–	–	0.282* n = 54
Headache severity	–	0.261* n = 57	–	–	–
HDI total	0.596** n = 57	0.581** n = 57	0.422** n = 57	0.476** n = 57	- 0.333* n = 54
Control					
Headache frequency	–	–	–	–	–
Headache severity	–	–	–	–	–
HDI total	–	–	–	–	–

*p < 0.05, ** p < 0.01
BES = Basic Emotions Scale, HDI = Headache Disability Inventory.
"-" = non-significant correlations

3.8.1.2. BES – Part 2 ("in general")

Table 3.22 shows the significant correlations found between the BES – Part 2 ("in general") and headache frequency, headache severity and HDI total for the two groups. In the headache group the HDI total score was found to be positively correlated with the experience of anger, sadness, disgust and fear "in general". A negative correlation was found between the HDI total score and the emotion of happiness. A positive correlation was found between the severity of headache and sadness. In the control group, the HDI total score was found to be positively

correlated with the experience of anger. No other correlations were found to be significant in the control group.

Table 3.22. Significant correlations between the BES – Part 2 ("in general") and headache frequency, headache severity and HDI total.

Measures by group	BES – Part 2 ("In general")				
	Anger	**Sadness**	**Disgust**	**Fear**	**Happiness**
Headache					
Headache frequency	_	_	_	_	_
Headache severity	_	0.299* n = 57	_	_	_
HDI total	0.632** n = 57	0.648** n = 57	0.379** n = 57	0.463** n = 57	- 0.307* n =57
Control					
Headache frequency	_	_	_	_	_
Headache severity	_	_	_	_	_
HDI total	0.311* n = 47	_	_	_	_

*p < 0.05, ** p < 0.01
BES = Basic Emotions Scale, HDI = Headache Disability Inventory.
"-" = non-significant correlations

3.8.1.3. BES – Part 3 ("coping")

Table 3.23 displays the significant correlations found between the BES – Part 3 ("coping") and headache frequency, headache severity and HDI total for the two groups. In the headache group the HDI total score was found to be positively correlated with the perceived inability to cope with the emotions of anger, sadness,

fear and happiness. In the control group, the severity of headache was found to be positively correlated with the perceived inability to cope with fear. No other correlations were found to be significant in either group.

Table 3.23. Significant correlations between the BES – Part 3 ("coping") and headache frequency, headache severity and HDI total.

Measures by group	BES – Part 3 ("Coping")				
	Anger	**Sadness**	**Disgust**	**Fear**	**Happiness**
Headache					
Headache frequency	–	–	–	–	–
Headache severity	–	–	–	–	–
HDI total	0.528** n = 47	0.528** n = 47	–	0.440** n = 47	0.376* n = 47
Control					
Headache frequency	–	–	–	–	–
Headache severity	–	–	–	0.321* n = 46	–
HDI total	–	–	–	–	–

*p < 0.05, ** p < 0.01
BES = Basic Emotions Scale, HDI = Headache Disability Inventory.
"-" = non-significant correlations

3.9. Emotion Regulation Questionnaire (ERQ)

3.9.1. ERQ- Part 1: General response to emotions

Table 3.24 shows the significant correlations found between the ERQ subscales and headache frequency, headache severity and HDI total for the two groups. In both groups the HDI total score was found to be positively correlated with the frequent use

of external-dysfunctional and internal-dysfunctional emotion regulation strategies. A positive correlation was found between the severity of headache and the frequent use of internal-dysfunctional strategies in the headache group. A positive correlation was found between the frequency of headache and the frequent use of external dysfunctional strategies in the control group. No other correlations were found to be significant.

Table 3.24. Significant correlations between the ERQ – Part 1 ("general response to emotions") and headache frequency, headache severity and HDI total.

Measures by group	ERQ – Part 1 ("general response to emotions)			
	Internal Dysfunctional	External Functional	Internal Functional	External Dysfunctional
Headache				
Headache frequency	–	–	–	–
Headache severity	0.349** n = 57	–	–	–
HDI total	0.592** n = 57	–	–	0.362** n = 57
Control				
Headache frequency	–	–	–	0.411** n = 47
Headache severity	–	–	–	–
HDI total	0.447** n = 47	–	–	0.481** n = 47

* $p < 0.05$, ** $p < 0.01$
ERQ = Emotion regulation Questionnaire
HDI = Headache Disability Inventory
"-" = non-significant correlations

3.9.2. ERQ- Part 2: General response to particular emotions

Pearson's correlation coefficients were calculated in order to examine the relationship between the frequent use of external-dysfunctional, internal-dysfunctional, external-functional and internal-functional emotion regulation strategies in relation to particular emotions (sadness, happiness, shame, anger, anxiety, guilt), and headache frequency, headache severity and HDI total for the two groups.

3.9.2.1. Sadness

Table 3.25 shows that in the headache group a positive correlation was found between the frequent use of internal- and external-dysfunctional strategies to regulate sadness and the HDI total score. In the control group the use of external-dysfunctional strategies was found to be positively correlated with the HDI total score. Also in the control group, a positive correlation was found between the frequency of headache and the frequent use of external-dysfunctional strategies to regulate sadness. No other correlations were found to be significant.

Table 3.25. Significant correlations between the ERQ – Part 2 ("sadness") and headache frequency, headache severity and HDI total.

Measures by group	ERQ (SADNESS)			
	Internal Dysfunctional	**External Functional**	**Internal Functional**	**External Dysfunctional**
Headache				
Headache frequency	_	_	_	_
Headache severity	_	_	_	_
HDI total	0.398** n = 57	_	_	0.339** n = 57
Control				
Headache frequency	_	_	_	0.402** n = 47
Headache severity	_	_	_	_
HDI total	_	_	_	0.311* n = 47

* $p < 0.05$, ** $p < 0.01$
ERQ = Emotion Regulation Questionnaire.
"-" = non-significant correlations

3.9.2.2. Happiness

Table 3.26 shows that in the headache group a positive correlation was found between the frequent use of internal-dysfunctional strategies to regulate happiness and the HDI total score. In the control group the use of external-functional strategies was found to be positively correlated with the frequency of headache. No other correlations were found to be significant.

Table 3.26. Significant correlations between the ERQ – Part 2 ("happiness") and headache frequency, headache severity and HDI total.

Measures by group	ERQ (HAPPINESS)			
	Internal Dysfunctional	External Functional	Internal Functional	External Dysfunctional
Headache				
Headache frequency	–	–	–	–
Headache severity	–	–	–	–
HDI total	0.291* n = 57	–	–	–
Control				
Headache frequency	–	0.289* n = 47	–	–
Headache severity	–	–	–	–
HDI total	–	–	–	–

* $p < 0.05$, ** $p < 0.01$
ERQ = Emotion Regulation Questionnaire.
"-" = non-significant correlations

3.9.2.3. Shame

Table 3.27 shows that in the headache group a positive correlation was found between the frequent use of external-dysfunctional strategies to regulate shame and the HDI total score. No other correlations were found to be significant in either group.

Table 3.27. Significant correlations between the ERQ – Part 2 ("shame") and headache frequency, headache severity and HDI total.

Measures by group	ERQ (SHAME)			
	Internal Dysfunctional	External Functional	Internal Functional	External Dysfunctional
Headache				
Headache frequency	–	–	–	–
Headache severity	–	–	–	–
HDI total	–	–	–	0.278* n = 57
Control				
Headache frequency	–	–	–	–
Headache severity	–	–	–	–
HDI total	–	–	–	–

* $p < 0.05$, ** $p < 0.01$
ERQ = Emotion Regulation Questionnaire.
"-" = non-significant correlations

3.9.2.4. Anger

Table 3.28 shows that in the headache group a positive correlation was found between the frequent use of external- and internal-dysfunctional strategies to regulate anger and the HDI total score. Also in the headache group, internal-functional strategies were found to be negatively correlated with the HDI total score. In the control group, external-dysfunctional strategies were found to be positively correlated with the HDI total score. No other correlations were found to be significant in either group.

Table 3.28. Significant correlations between the ERQ – Part 2 ("anger") and headache frequency, headache severity and HDI total.

Measures by group	ERQ (ANGER)			
	Internal Dysfunctional	External Functional	Internal Functional	External Dysfunctional
Headache				
Headache frequency	_	_	_	_
Headache severity	_	_	_	_
HDI total	0.301* n = 57	_	- 0.358** n = 57	0.325* n = 57
Control				
Headache frequency	_	_	_	_
Headache severity	_	_	_	_
HDI total	_	_	_	0.364* n = 46

* $p < 0.05$, ** $p < 0.01$
ERQ = Emotion Regulation Questionnaire.
"-" = non-significant correlations

3.9.2.5. Anxiety

Table 3.29 shows that in both groups a positive correlation was found between the frequent use of external-dysfunctional strategies to regulate anxiety and the HDI total score. In the headache group, internal-functional strategies were found to be negatively correlated with the HDI total score. In the control group, external-dysfunctional strategies were found to be positively correlated with the frequency of headache. No other correlations were found to be significant in either group.

Table 3.29. Significant correlations between the ERQ – Part 2 ("anxiety") and headache frequency, headache severity and HDI total.

Measures by group	ERQ (ANXIETY)			
	Internal Dysfunctional	External Functional	Internal Functional	External Dysfunctional
Headache				
Headache frequency	_	_	_	_
Headache severity	_	_	_	_
HDI total	_	_	- 0.269* n = 57	0.374** n = 57
Control				
Headache frequency	_	_	_	0.420** n = 46
Headache severity	_	_	_	_
HDI total	_	_	_	0.509** n = 46

* $p < 0.05$, ** $p < 0.01$
ERQ = Emotion Regulation Questionnaire.
"-" = non-significant correlations

3.9.2.6. Guilt

No significant correlations were found between the frequent use of external-dysfunctional, internal-dysfunctional, external-functional and internal-functional emotion regulation strategies in relation to guilt, and headache frequency, headache severity and HDI total in either group.

3.10. BDI-II, BAI, and STAXI.

Table 3.30 shows that in both groups a positive correlation was found between the BDI-II and BAI total scores and the HDI total score. In the control group only, a positive correlation was also found between the BAI total score and the severity of headache. In the headache group, all STAXI subscales were found to be positively correlated with the HDI total, with exception of the subscale Anger Control, which was found to be negatively correlated with the HDI total in both groups. In the control group, only the subscales Anger-Out and Anger Expression were found to be positively correlated with the HDI total. In the control group only, the subscale Anger-Out was found to be positively correlated with the severity of headache. No other correlations were found to be significant.

Table 3.30. Significant correlations between the BDI-II, BAI, STAXI, and headache frequency, headache severity and HDI total.

Measures by group	BDI-II	BAI	State Anger	Trait Anger	Trait-Temperament	Trait-Reactive	Anger-In	Anger-Out	Anger-Control	Anger Expression
Headache										
Headache frequency	–	–	–	–	–	–	–	–	–	–
Headache severity	–	–	–	–	–	–	–	–	–	–
HDI total	0.663** n = 55	0.490** n = 56	0.356** n = 56	0.510** n = 56	0.427** n = 56	0.337* n = 56	0.271* n = 56	0.340* n = 56	-0.395** n = 56	0.496** n = 56
Control										
Headache frequency	–	–	–	–	–	–	–	–	–	–
Headache severity	–	0.361* n = 46	–	–	–	–	–	0.420** n = 44	–	–
HDI total	0.424** n = 46	0.346* n = 46	–	–	–	–	–	0.552** n = 44	- 0.327* n = 44	0.584* n = 44

*p < 0.05, ** p < 0.01; "-" = non-significant correlations

3.11. Predicting headache severity and headache-related disability using multiple regression.

Using the stepwise method, multiple regression was employed to investigate whether the addition of anger-related variables contributed to predicting headache severity (NRS-101, VAS) and headache-related disability (HDI total) beyond that accounted for by depression and anxiety in the headache group. The following variables were entered in the analysis: "depression" (BDI-II), anxiety (BAI), "anger in general" (BES Part 2), "internal-dysfunctional" (ERQ subscale), "anger-in" (STAXI subscale), and "anger-ID" (ERQ, anger scale: internal-dysfunctional subscale)

3.11.1. Predictors of headache severity.

The frequent use of "internal-dysfunctional" emotion regulation strategies appeared as the only significant predictor of headache severity: Adjusted R square = 0.107, $F(1, 53) = 7.493$, $p < 0.01$ (see Table 3.31).

Table 3.31. Final model of multiple regression of BDI-II, BAI, "anger in general", "internal-dysfunctional" (ERQ subscale), "anger-in" (STAXI subscale), and "anger-ID" (ERQ, anger scale: internal-dysfunctional subscale), on headache severity.

Multiple Regression	**Dependent Variable = Headache Severity**	
Independent Variable	**β**	**p**
Internal Dysfunctional	0.352	0.008

3.11.2. Predictors of headache-related disability.

Model 1, which included only the BDI-II accounted for 42.9% of the variance (Adjusted R square = 0.429; F (1, 53) = 41.581, $p < 0.001$). The inclusion of "anger in general" into model 2 resulted in an additional 10% of the variance being explained (Adjusted R square = 0.522, F (2,52) = 30.483, $p < 0.001$). The final model 3 (Table 3.32) included "internal-dysfunctional", and this model accounted for 59.1 % of the variance (Adjusted R square = 0.591, F (3, 51) = 26.972, $p < 0.001$). Anxiety, anger-in, and anger-ID were not found to be significant predictors of headache-related disability.

Table 3.32. Final model of multiple regression of BDI-II, BAI, "anger in general", "internal-dysfunctional" (ERQ subscale), "anger-in" (STAXI subscale), and "anger-ID" (ERQ, anger scale: internal-dysfunctional subscale), on headache-related disability.

Multiple Regression	**Dependent Variable = Headache-related disability**	
Independent Variables	**β**	**p**
BDI-II	0.336	0.004
Anger in general	0.344	0.002
Internal Dysfunctional	0.309	0.003

4. Discussion

The aim of this study was to examine the emotional component of chronic headache and its association with headache severity and headache-related disability. A discussion of the main findings will be presented first, in the light of the literature reviewed in the Introduction, followed by their clinical implications. Next, the limitations of the study will be discussed, as well as possible avenues for future research.

4.1. Discussion of main findings in relation to psychological and headache measures.

4.1.1. Experience of basic emotions

One of the aims of this study was to identify which emotions are prominent amongst headache sufferers in comparison with non-headache controls. Results indicated that from a set of five basic emotions (anger, fear, disgust, sadness and happiness), the headache group was found to experience anger, fear and sadness more often than control participants, as predicted. These findings are consistent with the literature on chronic pain in general, and provide further support to the view that the affective component of pain incorporates a range of emotions that are primarily negative in nature (Fernandez & Milburn, 1994; Fernandez & Turk, 1995). Furthermore, these findings support the notion that pain is both sensation and emotion, as outlined in the definition of pain provided by the International Association for the Study of Pain (1986).

As predicted, a strong relationship was found between the experience of negative emotions and the presence of headache-related disability. Also, as predicted, the

emotion of happiness was found to be negatively correlated with headache-related disability. As for headache severity, only the emotion of sadness was found to be positively correlated with headache sufferers' pain intensity. Interestingly, a positive correlation was found between the frequency of headache and the emotion of happiness, though this was the case only for the experience of emotions within a recent period (state-like) rather than *in general* (trait-like).

Fernandez & Milburn (1994) argued that the emotion of happiness is the most likely to be dissociated from pain, as it is primarily a positive emotion. On the other hand, the emotions of anger, fear and sadness, are likely to be consistently linked to pain because they seem to be natural responses to aversive stimuli. The emotion of disgust is said to be more susceptible to cognitive modulation and therefore less associated with pain. This argument is based on the adaptive function of emotions, which presumes that when an organism is faced with an aversive event the adaptive responses available include fight, flight or submission, behaviours serviced by the emotions of anger, fear and sadness, respectively (Frijda, 1986; Plutchik, 1984). Thus, considering that pain is an aversive event, these emotions are likely to be triggered as part of an adaptive process.

According to Fernandez & Milburn (1994) the emotion of disgust seem to feature less prominently in pain perhaps because this emotion have less of a role in the adaptive response to aversive stimuli. In fact, irrespective of its link with pain, it has been observed that the emotion of disgust typically shows low reporting (Oatley & Duncan, 1992; Power, 2003), as found in this study.

Regarding the ability to cope with basic emotions, headache sufferers reported feeling less able to cope with virtually all emotions, including happiness. However, this difference was only significant in relation to the emotion of fear. A strong relationship was found between headache sufferers' inability to cope with the emotions of anger, sadness and fear, and the presence of headache-related disability.

No specific predictions were made in terms of how headache sufferers would differ from controls in terms of their ability to cope with basic emotions, as this type of investigation had not been done in previous studies. However, it has been repeatedly demonstrated that the use of ineffective coping skills to deal with chronic pain plays a significant role in maximizing the emotional and functional impacts of pain. For instance, the use of avoidant coping strategies – of which the emotion of fear is the main component – has been linked to pain severity, emotional distress and functional impairment (Holmes & Stevenson, 1990; Katz, Ritvo, Irvine & Jackson, 1996; Summerfeldt & Endler, 1998).

Interestingly, a positive correlation was found between the inability to cope with the emotion of happiness and headache-related disability. Even though the presence of a correlation does not imply causation, it could be argued that while the experience of positive emotions per se may help to decrease disability, the inability to cope with these emotions may result in an increase of disability. However, further systematic investigation would be needed to test out this assumption. In fact, while it has been demonstrated that negative emotional arousal is one of the most common precipitating factors in tension-type headache, migraine sufferers have indicated that any type of emotional arousal, including positive states such as feeling happy and excited, can

precipitate an attack (Donias, Peioglou-Harmoussi, Georgiadis & Manos, 1991). It has been argued that this is due to over-effective cognitive schemata processing, whereby any emotional arousal is interpreted as a dangerous signal, thus triggering off physiological mechanisms that result in the experience of pain. In a sense, this almost works as a protective mechanism, whereby pain alerts the organism of potential danger (Donias, Peioglou-Harmoussi, Georgiadis & Manos, 1991).

4.1.2. The regulation of emotions

Another aim of this study was to identify how headache sufferers regulate emotions and how this might differ from non-headache controls. As this aspect of the emotional component of headache had not yet been explored, no specific predictions were made. Results indicated that there were no significant differences between the headache group and the control group in styles of emotion regulation, although it was noted that headache sufferers reported using less internal- and external-functional strategies to regulate emotions *in general* than controls. Additionally, they indicated making more use of internal-dysfunctional strategies than controls, but less use of external-dysfunctional strategies.

As predicted, a positive correlation emerged between the frequent use of internal- and external-dysfunctional strategies and headache-related disability. Also, a positive correlation was observed between the use of internal-dysfunctional strategies and pain severity. The association between the use of dysfunctional strategies and perceived disability is consistent with previous findings where dysfunctional emotion regulation was found to be associated with psychosomatic complaints, including headache, and poorer subjective quality of life (Phillips, 2003).

The types of emotion regulation identified in the literature are characterised by either the inhibition or excessive expression of emotion (Garnefski, Kraaij & Spinhoven, 2001; Gross, 1999). The inhibition of emotion, in particular, has been linked to the aggravation of illnesses (Gross, 1989; Pennebaker, 1990). It is believed that the inhibition of emotions exacerbate physiological responses that may cause damage to physical health in the long term (Krantz & Manuck, 1984).

As discussed in the Introduction (section 1.4), dysfunctional emotion regulation involves a rejection or inhibition of emotion, whereas functional emotion regulation involves an acceptance of the emotion. Both styles can occur through the use of internal or external resources. In terms of individual differences, those who employ internal-functional regulation strategies use emotions as information that may indicate that cognitive change is necessary. Thus, they respond flexibly, accepting the information that the emotion conveys, in contrast to the emotion-rejecting manner of those who employ internal-dysfunctional strategies.

It has been argued that the type of emotional expression implicit in external-dysfunctional strategies (e.g. taking feelings out on other people or objects) may be seen as a complete lack of regulation, rather than a form of regulation (Southam-Gerow & Kendall, 2002). However, it is important to consider that this type of expression might have the purpose of alleviating an emotional state that the individual feels uncomfortable with. For instance, the emotion of anger is an unpleasant feeling that can be displaced onto others (Power & Dalgleish, 1997). Similarly, making others feel bad can be seen as a defensive attempt to protect the self from shame (Tangney, 1995). These strategies, however, are unlikely to alleviate the underlying

problem that generated the emotional state in first place. External-functional strategies (e.g. seeking social support) are also forms of emotional expression, however, they are likely to result in changes to the underlying problems that generated the emotion. Indeed, it has been argued that individuals who seek social support may become more aware of their emotional states (Ciarocchi, Scott, Deane & Heaven, 2003).

An examination of the emotion regulatory strategies employed in response to six particular emotions (sadness, happiness, shame, anger, anxiety, guilt) revealed that, the headache group reported making more use of internal ("I try not to let myself feel or express this") and external ("I take my feelings out on the people or objects around me") dysfunctional strategies, and less use of internal ("I change the way I view the situation") and external ("I seek social support") functional strategies than the control group. However, no significant differences were found between the two groups in relation to the experience of sadness, shame and guilt.

Regarding the emotion of anger, headache sufferers were found to employ internal-dysfunctional strategies significantly more often than controls, indicating that they usually try not to let themselves feel or express this emotion. This finding is in line with the literature on anger and pain, where the suppression of anger has been found to be strongly associated with chronic pain (Beutler, Engle, Oro'-Beutler, Daldrup & Meredith, 1986; Fernandez & Turk, 1995). The inhibition of angry feelings has been found to be the strongest predictor of reports of pain intensity and pain behaviour amongst other variables including depression, anger intensity and other styles of anger expression (Kerns, Rosenberg & Jacob, 1993). Studies comparing headache sufferers

with headache-free individuals have demonstrated that the former have significantly higher levels of trait anger and anger in (Arena, Bruno, Rozantine & Meador, 1997; Materazzo, Cathcart & Pritchard, 2000). Indeed, headache sufferers have been found to hold their anger in more than those without headache even after controlling for depression and anxiety (Nicholson, Gramling, Ong & Buenevar, 2003).

In relation to the emotion of anxiety, it was observed that the control group reported using external-functional regulation strategies significantly more often than the headache group, indicating that headache sufferers seek less social support when experiencing anxiety than controls. There are a number of possible explanations for this finding. While seeking social support involve sharing information about emotional experiences, which is primarily seen as an adaptive coping strategy, research has demonstrated that the role of social factors in chronic pain is somewhat double-edged. On one hand it has been demonstrated that social support is beneficial, as it helps pain sufferers to reduce levels of distress and inhibit avoidance behaviours, thus reducing disability (Cohen & Syme, 1985; Keefe, Smith, Buffington, Gibson, Studts & Caldwell, 2002). On the other hand, it has been postulated that support from significant others can be detrimental, as unhelpful pain behaviours may be positively reinforced by attention (Flor, Turk & Rudy, 1989). Therefore, even though seeking social support is regarded as a functional emotion regulation strategy, it is important to have in mind that no strategy can actually be seen as superior across all situations, as different strategies might have different consequences depending on the context (Gross, 1999a, Gross & Muñoz, 1995). This highlights the importance of understanding the advantages and disadvantages of diverse regulatory processes to help individuals match their style to situational demands, as it has been demonstrated

that different emotions might present different challenges in terms of emotion regulation strategies (Gross, 1998a, LeDoux, 1994).

Interestingly, the headache group was found to employ internal-functional strategies to regulate the emotion of happiness significantly more often than controls, suggesting that for headache sufferers positive emotions also require some degree of internal regulation, perhaps due to the effect of these feelings on the headache condition as already discussed.

4.1.3. Anger and anger expression styles as measured by the State-Trait Anger Expression Inventory (STAXI).

This study also aimed to examine the role of anger in chronic headache. While it was noted that headache sufferers scored higher in all measures of anger (State-Anger and Trait-Anger) and anger expression style (Anger-in, Anger-out, Anger Control) than controls, the difference between the groups was not significant. Therefore the hypothesis that headache sufferers would present higher levels of trait anger and anger-in, as measured by the STAXI, was not supported.

It could be argued that a bigger sample size could have made a difference in this comparison, however, a couple of recent studies using different sample sizes have found significant differences between headache and non-headache individuals in at least two of the STAXI subscales. One study compared tension-type headache sufferers (n = 16) and migraine sufferers (n = 28) with headache-free controls (n = 38). Results indicated that while the tension-type headache group had significantly higher levels of anger expression and anger-in than controls, the difference between

migraineurs and controls was not significant. Nonetheless, both of the headache groups did have non-significantly higher levels of anger than controls (Matterazzo, Cathcart & Pritchard, 2000). A more recent study using a sample of 422 participants found that although headache participants scored significantly higher on trait anger and anger-in than non-headache controls, the effect was relatively small, with anger-in being the highest (Nicholson, Gramling, Ong & Buenevar, 2003).

One important factor distinguishing this study from these two mentioned is that the lack of significant differences may reflect the nature of the sample used. This study used a clinical sample of headache sufferers, whereas the two studies mentioned above used headache sufferers from the general community. Additionally, it has been documented that headache patients have a tendency to deny feelings of anger and aggressiveness in comparison with pain-free controls (Franz, Paul, Bautz, Choroba & Hildelbrandt, 1986). In fact, in this study it was demonstrated that headache sufferers differ significantly from controls in their tendency to suppress the feeling and expression of anger, as measured by the internal-dysfunctional subscale in relation to the emotion of anger.

Interestingly, headache sufferers differed significantly from controls in their report of the experience of anger both within a recent time frame (state-like) and in general (trait-like), as measured by the Basic Emotions Scale. This raises some issues about what exactly an individual recalls when making a judgement about the frequency with which emotion states are experienced. Of particular relevance is the idiosyncratic connotation an emotion term has for individual users of the language (Power, 2003). Therefore, it might be the case that the items of the STAXI subscales examining state

and trait anger were loaded with meanings that did not reflect the experience of this sample of headache sufferers, whereas in the Basic Emotions Scale no meaning was imposed and therefore the results reflected the idiosyncratic meaning attributed to the emotion of anger.

In the headache group, all STAXI subscales were found to be positively correlated with headache-related disability, with exception of the subscale Anger Control, which was found to be negatively correlated with disability in both groups. This suggests that the presence of anger, as well as an anger management style characterised by suppression and aggression, is associated with the presence of headache-related disability, as already discussed. On the other hand, exerting control over the emotion of anger appears to be associated with experiencing less disability. However, it has been argued that while anger control is generally viewed as being positive, overly high levels of anger control can actually reduce the likelihood of appropriate expression of anger, which can also be detrimental for well being (Greenwood, Thurston, Rumble, Waters & Keefe, 2003).

4.1.4. Depression and Anxiety

As predicted, the headache group showed significantly higher levels of depression and anxiety, as measured by the BDI-II and BAI, than the control group, suggesting that the experience of chronic headache may affect psychological well being. Also as predicted, a positive correlation was found between depression and anxiety symptoms and headache-related disability.

Psychological conditions such as anxiety and depression appear to be high amongst headache sufferers (e.g. Breslau, Merikangas & Bowden, 1994; Holroyd, Stensland, Lipchik, Hill, O'Donnel & Cordiney, 2000). In this study, the findings that the emotions of fear and sadness are prominent emotions amongst headache sufferers is consistent with the higher levels of depression and anxiety symptoms found amongst them.

The cause of psychological distress in headache sufferers remains controversial. One of the main issues in the literature concerns the relative importance of trait versus state influences. Some have argued that it is not the headache that leads to emotional disturbance but preexisting emotional distress and psychological vulnerability (Turk & Salovery, 1984). Others believe that emotional disturbance results from the discomfort of the headache itself rather than the premorbid vulnerability (Gaskin, Greene, Robinson & Geisser, 1992). Still, there are others who associate the psychological distress with the effects of general life changes and stressors rather than with the experience of headache itself (Wise, Mann, Jani & Jani, 1994).

4.1.5. Predictors of headache severity and headache-related disability

It was hypothesised that the addition of anger-related variables to a model consisting of depression and anxiety would contribute significantly to a model predicting headache severity and headache-related disability. In relation to headache severity, results offered very little support for this hypothesis, since the frequent use of an internal-dysfunctional style of emotion regulation was the only predictor of headache severity. Interestingly, in this study the intensity of headache was far less associated with psychological disturbance in general, than headache-related disability. The

frequency of headache was even less associated with emotional disturbance. It is possible that once headache becomes a chronic condition, the frequency and severity of pain have less impact on affective states than headache-related emotional and functional disability, as it affects quality of life directly.

The main predictors of headache-related disability were depression (BDI-II), the frequent experience of anger ("anger in general"), and the frequent use of internal-dysfunctional strategies to regulate emotions. These results offer only partial support to the hypothesis above because anxiety did not come across as a significant predictor of disability.

4.2. Clinical implications

The findings of this study concurs with previous research in suggesting that there is an association between negative affect and headache. While various multidimensional models of pain and emotion have been proposed, as discussed in the Introduction (Section 1.5), the clinical application of these models require an understanding of the relative contribution of specific affective dimensions to the subjective experience of pain. An investigation of the type of negative emotions prominent amongst headache sufferers is an essential element of this process. The findings that the emotions of anger, sadness, and fear are more prominent in headache sufferers than in headache-free individuals highlight the need for treatment that is targeted at the individual as a whole person and not only at the pain itself. This is particularly relevant because, while a specific treatment may provide some pain relief, the failure to comprehend the emotional focus of the patient's suffering can actually increase the total suffering (Cassel, 1991). For example, it has been demonstrated that headache sufferers are at

increased risk of developing depression (Breslau, Schultz, Stewart, Lipton, Lucia & Welch, 2000). This has important implications for the management of headaches, as it has been demonstrated that depressed headache sufferers have poorer outcome on follow-up when compared with non-depressed headache sufferers (Curioso, Young, Schechter & Kaiser, 1999; Devlen, 1994; Mitsikostas & Thomas, 1999).

Psychological intervention for pain management often address anxiety and depression issues through the teaching of anxiety management techniques and the role of negative thoughts and beliefs about pain on mood (Martin, 1993). However, the effect of the emotion of anger on pain is often overlooked (Fernandez & Turk, 1995). It has been suggested that high levels of anger leads to an increase in skeletal muscle tension resulting in pain (Burns, 1997). As previously mentioned, the inhibition of angry feelings has been found to be the strongest predictor of reports of pain intensity and pain behaviour amongst other variables including depression, anger intensity and other styles of anger expression (Kerns, Rosenberg & Jacob, 1993).

In addition to exacerbating pain, there is evidence that anger and anger management styles can affect the treatment outcome of chronic pain sufferers. For instance, it has been demonstrated that patients with higher levels of anger suppression show less improvement following cognitive behavioural intervention for pain (Burns, Johnson, Devine, Mahoney & Pawl, 1998). Fernandez and Turk (1995) argued that pain patients' manifestation of anger can disrupt relationships with healthcare providers, thus undermining the attainment of treatment goals. Moreover, in pain management groups angry patients may potentially disrupt the group process, hence the importance of targeting anger-related variables for modification in treatment.

This study has provided some evidence to suggest that the use of dysfunctional emotion regulation strategies, particularly regarding the emotions of anger and anxiety, is associated with perceived headache-disability. Considering that emotion regulation affects the emotional states experienced (Gross, 1999), this highlights the importance of applying emotion regulation models to the psychological treatment of headaches. Identifying the emotion regulation strategies used by headache sufferers may facilitate the development of psychological treatment targeted at reducing the emotional and functional disabling effect of headache.

4.3. Limitations of study

4.3.1. Limitations in design

The design of the study relied upon the self-report and recall of affective states. This type of limitation affects most studies on emotion, as most of what is known about the subjective experience of emotion comes from questionnaires filled out by individuals who are trying to remember what a given emotion feels like (Ekman, 1999). Additionally, self-reported evaluations may be an under- or over-estimation of the extent to which the subjective experience occurred in reality.

The study did not take into account the current use of medication, therefore the mediating effect of symptomatic and prophylactic medications on affective distress was not evaluated. It is possible that those patients who were benefiting from medication reported lower levels of emotional disturbance, than those for whom treatment has been ineffective.

The headache sample was selected from a clinical setting and therefore the findings may only characterise those individuals who seek treatment, and may constitute a biased sample in terms of exaggerated psychopathology.

No qualitative data was obtained either through interviews or questionnaire due to time constraints. In particular, this type of data would have been useful to clarify the idiosyncratic meaning respondents attributed to emotion terms.

4.3.2. Limitations of measures

The Basic Emotions Scale relies on the idiosyncratic meaning individuals attribute to each specific emotion. While this may be advantageous, as already argued, it means that the subjective experience the individual recalled and associated with a certain emotion remains unknown.

To the author's knowledge this study was the first to examine the emotion regulation style of chronic headache sufferers. The field of emotion regulation is relatively new, therefore measures designed to evaluate individual differences in emotion regulation styles are still being developed. The Emotion Regulation Questionnaire was standardised within a population of normal adolescents which explains why some of the items used to evaluate external-dysfunctional strategies reflect behaviours that are characteristic amongst young people such as "I take my feelings out on other people or objects around me" (e.g. lashing out, bullying, arguing). This may account for the low reporting in the use of dysfunctional strategies. The main advantage of using this measure was that it had been used to evaluate the association between emotion

regulation styles with psychosomatic complaints, including headache, and epilepsy, a long-term illness.

Some of the Headache Disability Index (HDI) items are anger related (e.g. "My headaches make me angry", "Sometimes I feel that I'm going to lose control because of my headaches", "I feel irritable because of my headaches") and it is possible that the significant correlations found between the HDI and the other measures of anger could be partly due to the presence of redundant information within the scales.

4.4. Future research

While this study provided some insight into the emotion regulation styles of headache sufferers, in comparison with headache-free individuals, this issue needs to be further explored. For example, it would be interesting to know how the type of headache condition may affect the experience and regulation of emotions. Some studies have separately examined the differences between migraineurs and tension-type headache sufferers, against headache-free individuals, on measures of anger, depression, and coping strategies and found significant differences between the three groups (e.g. Materazzo, Cathcart & Pritchard, 2000). It would also be interesting to investigate further the role of the emotion of happiness on chronic headache, perhaps through a mix of quantitative and qualitative methods.

It could be relevant to explore whether there are gender differences in the experience and regulation of emotions, as it is the case regarding coping strategies (Rollnik, Karst, Piepenbrock, Gehrke, Dengler & Fink, 2003; Unruh, 1996). This would probably require a more balanced number of male/female participants, which is

uncommon in most studies, as females are more prone to most headache disorders and often outnumber males in research.

The sample studied seems to be representative of the headache population in several aspects as outlined in the Introduction (Section 1.6). For instance, a female preponderance was found, with migraine and tension-type headaches being the most common types of headache. Participants were of a wide age range and had been suffering from chronic headaches for a considerable number of years. Patients experienced a considerable amount of disability due to their headaches and presented higher symptoms of depression and anxiety when compared to controls, although these did not fall in the clinical range for the majority of headache participants, which concurs with previous studies (e.g. Nicholson, Gramling, Ong & Buenevar, 2003). However, due to the number and type of measures used in the study, a high number of statistical comparisons were carried out. This has inevitably increased the risk of significant values being found purely by chance and, therefore, these should be interpreted with caution. Replication of the study is desirable to evaluate whether the findings are consistent across different samples.

4.5. Conclusions

In this study it was found that headache sufferers presented higher levels of emotional disturbance than headache-free individuals. For instance, the basic emotions of anger, sadness and fear were found to be experienced more often by headache sufferers than controls. Headache sufferers also reported feeling less able to cope with the emotion of fear, which is the main component of anxiety. Additionally, headache sufferers showed greater use of dysfunctional strategies to regulate the

emotion of anger, and less use of functional strategies to regulate anxiety. Accordingly, higher levels of anxiety and depression symptoms were found amongst headache sufferers.

Affective distress was found to be associated with perceived emotional and functional headache-related disability and, to a lesser extent, with headache severity. Indeed, the presence of depression symptoms, anger, and the use of internal-dysfunctional strategies were found to be significant predictors of headache-related disability. Furthermore, the use of internal-dysfunctional emotion regulation strategies was found to be a significant predictor of headache severity. These findings suggest that the sensory and affective dimensions of pain are amalgamated and deserve equal priority for treatment.

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Appendix I

LOTHIAN RESEARCH ETHICS COMMITTEE

CERTIFICATE OF ETHICAL OPINION

LREC Reference Number: LREC/2003/7/40
Title: The emotional component of chronic headache and its relationship to perceived pain severity and disability.
Researcher: Mrs Norma Martin

The Primary Care/Public & Mental Health Research Ethics Committee of the Lothian Research Ethics Committee (the Committee) reviewed this proposed research and is of the opinion that it is ethical and appropriate to be carried out in the Lothian Area. This opinion encompasses all aspects of the application including the Patient/Subject Information Sheet and all other accompanying documentation provided.

The LREC application form, protocol, subject information sheet, information on compensation arrangements, payments to researchers and the provision of expenses to subjects (where appropriate) were reviewed and approved and the members of the Committee present at the meeting are shown on the attached *Membership List*.

This opinion is issued subject to the following conditions and is invalid if they are not followed:

- You must obtain appropriate management approval from the relevant NHS Trust(s) before starting the proposed research. It is the NHS Trust(s) that ultimately decide whether or not this research should go ahead taking account of the advice of the Local Research Ethics Committee.
- You must notify the Sub-Committee and the relevant NHS Trust(s), in advance, of any significant proposed deviation from the original protocol or application form and obtain approval for any such amendments using the *Amendment Approval Request Form*.
- You must submit reports to the Sub-Committee and the NHS Trust(s) once the study is underway if there are any unusual or unexpected results which raise questions about the safety of the research.
- You must report annually on successes, or difficulties, in recruiting subjects in order to provide useful feedback on perceptions of the study among patients and volunteers using the *Progress Report Form*.
- Where the study is terminated prematurely you must report within fifteen days indicating the reasons for early termination.
- You must submit a final report within three months of the completion of the study using the *Progress Report Form*.
- This opinion does not cover the inclusions of adults with incapacity in any study. Such opinion can only be given by the Multi-Centre Research Ethics Committee for Scotland.

Peter Reith
Secretary
Lothian Research Ethics Committee

Stephanie Butler
Administrator
Primary Care/Public & Mental Health
Research Ethics Committee

26 January 2004

Lothian Primary Care NHS Trust

Lrec 2003/7/40
Our Ref: JEW/AMH/03/089
Enquiries to: Jackie Warburton
Ext. No: 0131 537 9522
Date: 28 January 2004

Headquarters
St Roque
Astley Ainslie Hospital
133 Grange Loan
Edinburgh EH9 2HL
Telephone 0131 537 9000
Reception 0131 537 9525
Fax 0131 537 9500
www.show.scot.nhs.uk/lpct/

NHS
Lothian

Mrs Norma Martin
Dept of Psychology
40 Colinton Road
EDINBURGH
EH10 5BT

Dear Mrs Martin

Research Proposal: THE EMOTIONAL COMPONENT OF CHRONIC HEADACHE AND ITS RELATIONSHIP TO PERCEIVED PAIN SEVERITY AND DISABILITY

I refer to your recent application to Lothian Primary Care NHS Trust. This has now been considered by the Trust's Research and Development Committee and I would like to confirm that the Lothian Primary Care NHS Trust approves your proposal subject to the written approval of the Research Ethics Sub-Committee being obtained prior to commencement of the study. Your proposal will also require Lothian University Hospitals Operational Division approval and therefore the Trust's Research and Development Manager has forwarded a copy to their Research and Development office for consideration.

A condition of this approval is that you advise me, in advance, of any significant proposed deviation from the original protocol including significant changes to the dates when this research will be active.

I would like to remind you that research must be conducted in accordance with the research governance framework and I enclose a copy of the responsibilities of the Principal Investigator extracted from the framework. One of the conditions of the framework is that any researchers who have access to patients, patient data or records and who are not employed by the NHS must have an honorary NHS contract. If you need to arrange this please contact the Trust Research Manager.

Details of your research will be forwarded to the National Research Register in about six months time. Therefore, if for any reason this research does not go ahead I would be grateful if you could advise me.

With best wishes.

Yours sincerely

MURRAY DUNCANSON
CHIEF EXECUTIVE
cc Stephanie Butler, Simon Fawcett

The Lothian University
Hospitals NHS Trust

NHS
Lothian

ROYAL INFIRMARY OF EDINBURGH
51 Little France Crescent, Edinburgh, EH16 4SA

HAC/KS/app1a/patnoncomm

20 February 2004

Dr R Cull
Clinical Neurophysiology
Western General Hospital
Crewe Road
Edinburgh

Dear Dr Cull

LREC No: **2003/7/40**
R&D Project ID No: **2003/W/PSY/02**
Title of Research: *The emotional component of chronic headache and its relationship to perceived pain severity and disability*

The above project has undergone a review of resource and financial implications by the R&D Office and I am satisfied that all the necessary arrangements have been set in place.

On behalf of the Chief Executive and Medical Director, I am happy to give division management approval to allow the project to commence, subject to the approval of the appropriate Research Ethics Sub-Committee having also been obtained.

We would ask you to note that under Section 7, question 34, NHS Lothian provides indemnity for negligence for NHS and honorary clinical staff wherever research involves patients attending the hospitals. It is not empowered to provide non negligent indemnity for patients or volunteers.

Yours sincerely

Dr Heather A Cubie
R&D Director

cc *Secretary, Research Ethics Sub-Committee*
Mrs N MARTIN, Clinical PSYCHOLOGY

RESEARCH & DEVELOPMENT OFFICE

Tel: 0131 242 3330
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Mrs Patricia Murray
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Assistant Accountant:
Neil Mclean

Commercial Research Administrator
Miss Sara Lindsay
BSc (Hons)

Administrative Assistant:
Miss Kellyann Stewart
Mr Neil Feltham

Appendix II

NHS Lothian

19th December 2003

Participant Information Sheet

Emotions and Chronic Headache

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with friends, relatives and your GP if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

What is the purpose of the study?

The study is being carried out in order to increase our understanding of how emotions are associated with the experience of chronic headaches. We would like to investigate what types of emotions chronic headache sufferers experience more often, how they cope with those emotions, and how their experience may differ from pain-free individuals. In addition we would like to investigate how negative emotions are related to the severity of headaches and to psychological and social well being. In furthering our understanding, we hope to be in a better position to offer effective and appropriate help.

What will I have to do?

If you decide to take part, you will be asked to complete a series of questionnaires. These questionnaires will ask about your experience of various emotions (such as happiness, sadness, fear and anger) and mood states (such as anxiety and depression). They will also ask how you generally deal with these emotions. In addition you will be asked about the severity of your pain and how this affects your life. It will take approximately 1 hour to take part in the study.

Why have I been asked to take part?

The study is running from January 2004 to June 2004. People being referred to the Headache Clinic at the Western General Hospital, and to the pain management programme at the Clinical Psychology Department of the Royal Edinburgh Hospital, during this time are being asked to take part.

Will my participation be kept confidential?

All information collected as part of the research will be kept strictly confidential and subject to the Data Protection Act. Any information will have your name and address removed so that you cannot be recognised from it. If participation in the study raises any issues for you, the researcher will be happy to discuss these with you and, with

your consent, share any concerns with your doctor or therapist. If you wish your General Practitioner will receive notification that you are taking part in this study.

Who is organising the research?

The research is being carried out by Norma Martin (Trainee Clinical Psychologist) as part of the University of Edinburgh Clinical Psychology training course requirements. She is being supervised by Dr Alison Harper (Clinical Psychologist) at the Clinical Psychology Department of the Royal Edinburgh Hospital. This study has been reviewed by the relevant research ethics committee in Lothian.

What happens if I decide to take part?

If you decide to take part, please complete the slip provided, sign the consent form, and send both of these in the envelope provided. You will then be contacted by the lead researcher, Norma Martin, and an appointment will be arranged.

What happens if I decide not to take part?

Taking part in this study is completely voluntary. If you decide to take part you are still free to withdraw at any time without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect the standard of care you receive.

Local Independent Advisor

If required, you can contact Dr Paul Morris, Lecturer in Health Psychology, as an independent advisor, to discuss any questions you may have about the research. he can be contacted at the following address:

Dr Paul Morris
Lecturer in Health Psychology
Kennedy Tower
Royal Edinburgh Hospital
Edinburgh
EH10 5HF. Telephone (0131) 537 6279

Thank you for reading this and for your consideration.

Patient Identification Number:

CONSENT FORM

Title of Project: Emotions and Chronic Headache.

Name of researcher: Norma Martin, Trainee Clinical Psychologist.

Please initial

I confirm that I have read and understood the information sheet dated 19th December 2003 for the above study and had the opportunity to ask questions. _____

I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. _____

I understand that sections of any of my medical notes may be looked at by responsible individuals from regulatory authorities, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records. _____

I agree to take part in the above study. _____

_____	_____	_____
Name of Patient	Date	Signature
_____	_____	_____
Researcher	Date	Signature

1 for patient; 1 for researcher; 1 to be kept with hospital notes

NHS Lothian

19th December 2003

Participant Information Sheet

Emotions and Chronic Headache

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

What is the purpose of the study?

The study is being carried out in order to increase our understanding of how emotions are associated with the experience of chronic headaches. We would like to investigate what types of emotions chronic headache sufferers experience more often, how they cope with those emotions, and how their experience may differ from pain-free individuals. In addition we would like to investigate how negative emotions are related to the severity of headaches and to psychological and social well being. In furthering our understanding, we hope to be in a better position to offer effective and appropriate help.

What will I have to do?

If you decide to take part, you will be asked to complete a series of questionnaires. These questionnaires will ask about your experience of various emotions (such as happiness, sadness, fear and anger) and mood states (such as anxiety and depression). They will also ask how you generally deal with these emotions. In addition you will be asked about the severity of your pain and how this affects your life. It will take approximately 1 hour to take part in the study.

Why have I been asked to take part?

The study is running from January 2004 to June 2004. People suffering from recurrent headache attending appointments at the Headache Clinic (Western General Hospital) and at the Clinical Psychology Department (Royal Edinburgh Hospital), during this time are being asked to take part. In order to look at differences between the emotional experiences of people who experience chronic headache and those who do not, a comparison group of people who do not suffer from chronic headaches have been asked to consider participating. You have been asked to participate in this comparison group of non-headache sufferers.

Headquarters:
St. Roque, Astley Ainslie Hospital, 133 Grange Loan, Edinburgh EH9 2HL

Chairman Garth Morrison CBE
Chief Executive Murray Duncanson

Will my participation be kept confidential?

All information collected as part of the research will be kept strictly confidential. Any information will have your name removed so that you cannot be recognised from it.

Who is organising the research?

The research is being carried out by Norma Martin (Trainee Clinical Psychologist) as part of the University of Edinburgh Clinical Psychology training course requirements. She is being supervised by Dr Alison Harper (Clinical Psychologist) at the Clinical Psychology Department of the Royal Edinburgh Hospital. This study has been reviewed by the relevant research ethics committee in Lothian.

What happens if I decide to take part?

If you decide to take part, please sign the consent form, complete the questionnaire package and send both in the stamped addressed envelope provided.

What happens if I decide not to take part?

Taking part in this study is completely voluntary. If you decide to take part you are still free to withdraw at any time without giving a reason.

Local Independent Advisor

If required, you can contact Dr Paul Morris, Lecturer in Health Psychology, as an independent advisor, to discuss any questions you may have about the research. He can be contacted at the following address:

Dr Paul Morris
Lecturer in Health Psychology
Kennedy Tower
Royal Edinburgh Hospital
Edinburgh
EH10 5HF. Telephone (0131) 537 6279

Thank you for reading this and for your consideration.

Identification Number:

CONSENT FORM

Title of Project: Emotions and Chronic Headache.

Name of researcher: Norma Martin, Trainee Clinical Psychologist.

Please initial

I confirm that I have read and understood the information sheet dated 19th December 2003 for the above study and had the opportunity to ask questions. _____

I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my legal rights being affected. _____

I agree to take part in the above study. _____

_____	_____	_____
Name of Participant	Date	Signature
_____	_____	_____
Researcher	Date	Signature

1 for participant; 1 for researcher

Appendix III

DEMOGRAPHIC COVER SHEET

Name _____ Sex_____Age _____ Date _____

Education_____ Occupation _____ Marital Status _____

1. How long have you had chronic headache?

.....Years ..... Months

2. What type of chronic headache do you experience? Please ✓

Migraine ..... ☐

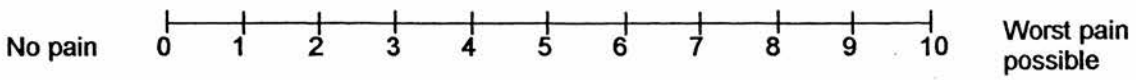
Tension-type..... ☐

Combined migraine/tension-type..... ☐

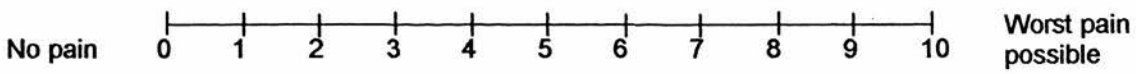
Cluster type ..... ☐

Other (please specify)..... ☐

4. Please indicate on the line below the level of your pain, currently.



5. Please indicate on the line below the level of your pain on average.



HEADACHE DISABILITY INDEX

Patient Name _____ Date: _____

INSTRUCTIONS: Please CIRCLE the correct response:
1. I have headache: (1) 1 per month (2) more than 1 but less than 4 per month (3) more than one per week

2. My headache is: (1) mild (2) moderate (3) severe

Please read carefully: The purpose of the scale is to identify difficulties that you may be experiencing because of your headache. Please CIRCLE "YES", "SOMETIMES" or "NO" to each item. Answer each question as it pertains to your headache only.

YES SOMETIMES NO **E1. Because of my headaches I feel handicapped.**

YES SOMETIMES NO **F2. Because of my headaches I feel restricted in performing my routine daily activities.**

YES SOMETIMES NO **E3. No one understands the effect my headaches have on my life.**

YES SOMETIMES NO **F4. I restrict my recreational activities (e.g. sports, hobbies) because of my headaches.**

YES SOMETIMES NO **E5. My headaches make me angry.**

YES SOMETIMES NO **E6. Sometimes I feel that I am going to lose control because of my headaches.**

YES SOMETIMES NO **F7. Because of my headaches I am less likely to socialise.**

YES SOMETIMES NO **E8. My spouse (significant other), or family and friends have no idea what I am going through because of my headaches.**

YES SOMETIMES NO **E9. My headaches are so bad that I feel that I am going to go insane.**

YES SOMETIMES NO **E10. My outlook on the world is affected by my headaches.**

YES SOMETIMES NO **E11. I am afraid to go outside when I feel that a headache is starting.**

YES SOMETIMES NO **E12. I feel desperate because of my headaches.**

YES SOMETIMES NO **F13. I am concerned that I am paying penalties at work or at home because of my headaches.**

YES SOMETIMES NO **F14. My headaches place stress on my relationships with family or friends.**

YES SOMETIMES NO **F15. I avoid being around people when I have a headache.**

YES SOMETIMES NO **F16. I believe my headaches are making it difficult for me to achieve my goals in life.**

YES SOMETIMES NO **F17. I am unable to think clearly because of my headaches.**

YES SOMETIMES NO **F18. I get tense (e.g. muscle tension) because of my headaches.**

YES SOMETIMES NO **F19. I do not enjoy social gatherings because of my headaches.**

YES SOMETIMES NO **E20. I feel irritable because of my headaches.**

YES SOMETIMES NO **F21. I avoid travelling because of my headaches.**

YES SOMETIMES NO **E22. My headaches make me feel confused.**

YES SOMETIMES NO **E23. My headaches make me feel frustrated.**

YES SOMETIMES NO **F24. I find it difficult to read because of my headaches.**

YES SOMETIMES NO **F25. I find it difficult to focus my attention away from my headaches and on other things.**

THE BASIC EMOTIONS SCALE

The purpose of this scale is to find out about how much or how often you experience certain emotions and then to ask some questions about how you feel actually during particular emotions themselves.

The first part of the scale is designed to explore how you have felt **DURING THE LAST WEEK.**
For each emotion, please circle **ONE** number only between 1 and 7, to indicate how you have felt.

OVER THE PAST WEEK I HAVE FELT :

	not at all			**some of the time**			**all of the time**
ANGER	1	2	3	4	5	6	7
DESPAIR	1	2	3	4	5	6	7
SHAME	1	2	3	4	5	6	7
ANXIETY	1	2	3	4	5	6	7
HAPPINESS	1	2	3	4	5	6	7
FRUSTRATION	1	2	3	4	5	6	7
MISERY	1	2	3	4	5	6	7
GUILT	1	2	3	4	5	6	7
NERVOUSNESS	1	2	3	4	5	6	7
JOY	1	2	3	4	5	6	7
IRRITATION	1	2	3	4	5	6	7
GLOOMINESS	1	2	3	4	5	6	7
HUMILIATED	1	2	3	4	5	6	7
TENSE	1	2	3	4	5	6	7
LOVING	1	2	3	4	5	6	7
AGGRESSION	1	2	3	4	5	6	7
MOURNFUL	1	2	3	4	5	6	7
BLAMEWORTHY	1	2	3	4	5	6	7
WORRIED	1	2	3	4	5	6	7
CHEERFUL	1	2	3	4	5	6	7

In the second part of this questionnaire we would like to know about how you feel **IN GENERAL.**

The question asks about **HOW OFTEN** you feel the emotion.

Again, for each question please circle **ONE** number only between 1 and 7 to indicate how you feel.

IN GENERAL , I FEEL THIS EMOTION :

	never			sometimes			very often
ANGER	1	2	3	4	5	6	7
DESPAIR	1	2	3	4	5	6	7
SHAME	1	2	3	4	5	6	7
ANXIETY	1	2	3	4	5	6	7
HAPPINESS	1	2	3	4	5	6	7
FRUSTRATION	1	2	3	4	5	6	7
MISERY	1	2	3	4	5	6	7
GUILT	1	2	3	4	5	6	7
NERVOUSNESS	1	2	3	4	5	6	7
JOY	1	2	3	4	5	6	7
IRRITATION	1	2	3	4	5	6	7
GLOOMINESS	1	2	3	4	5	6	7
HUMILIATED	1	2	3	4	5	6	7
TENSE	1	2	3	4	5	6	7
LOVING	1	2	3	4	5	6	7
AGGRESSION	1	2	3	4	5	6	7
MOURNFUL	1	2	3	4	5	6	7
BLAMEWORTHY	1	2	3	4	5	6	7
WORRIED	1	2	3	4	5	6	7
CHEERFUL	1	2	3	4	5	6	7

In the third part of this questionnaire we would like to ask you for some information about **HOW WELL YOU FEEL YOU COPE** when you experience that emotion. For example, you might feel completely out of control of the emotion, or overwhelmed by the emotion in some other way.

Please note: even if you **never** experience a particular emotion, please answer the question by imagining how you think you would feel if you did experience that emotion.

Again, for each part of the question, please circle **ONE** number between 1 and 7 to indicate how well you feel you cope with the emotion.

	Cope very well						**Cope very badly**
ANGER	1	2	3	4	5	6	7
DESPAIR	1	2	3	4	5	6	7
SHAME	1	2	3	4	5	6	7
ANXIETY	1	2	3	4	5	6	7
HAPPINESS	1	2	3	4	5	6	7
FRUSTRATION	1	2	3	4	5	6	7
MISERY	1	2	3	4	5	6	7
GUILT	1	2	3	4	5	6	7
NERVOUSNESS	1	2	3	4	5	6	7
JOY	1	2	3	4	5	6	7
IRRITATION	1	2	3	4	5	6	7
GLOOMINESS	1	2	3	4	5	6	7
HUMILIATED	1	2	3	4	5	6	7
TENSE	1	2	3	4	5	6	7
LOVING	1	2	3	4	5	6	7
AGGRESSION	1	2	3	4	5	6	7
MOURNFUL	1	2	3	4	5	6	7
BLAMEWORTHY	1	2	3	4	5	6	7
WORRIED	1	2	3	4	5	6	7
CHEERFUL	1	2	3	4	5	6	7

Thank You Very Much For Your Help With This Questionnaire

How You Respond To Your Emotions…

PART A

We all experience lots of different feelings or emotions. For example, different things in our lives make us feel happy, sad, angry and so on…

The following questions ask you to think about **how often** you do certain things **in response to your emotions**. You do not have to think about specific emotions but just how often you **generally** do the things listed below.

We all respond to our emotions in different ways so there are no right or wrong answers.

In GENERAL how do you respond to your emotions?	Never	Seldom	Often	Very Often	Always
1. I talk to someone about how I feel	O	O	O	O	O
2. I take my feelings out on others verbally (e.g. shouting, arguing)	O	O	O	O	O
3. I seek physical contact from friends or family (e.g. a hug, hold hands)	O	O	O	O	O
4. I review (rethink) my thoughts or beliefs	O	O	O	O	O
5. I harm or punish myself in some way	O	O	O	O	O
6. I do something energetic (e.g. play sport, go for a walk)	O	O	O	O	O
7. I dwell on my thoughts and feelings (e.g. It goes round and round in my head and I can't stop it)	O	O	O	O	O
8. I ask others for advice	O	O	O	O	O
9. I review (rethink) my goals or plans	O	O	O	O	O

In GENERAL how do you respond to your emotions?	Never	Seldom	Often	Very Often	Always
10. I take my feelings out on others physically (e.g. fighting, lashing out)	O	O	O	O	O
11. I put the situation into perspective	O	O	O	O	O
12. I concentrate on a pleasant activity	O	O	O	O	O
13. I try to make others feel bad (e.g. being rude, ignoring them)	O	O	O	O	O
14. I think about people better off and make myself feel worse	O	O	O	O	O
15. I plan what I could do better next time	O	O	O	O	O
16. I keep the feeling locked up inside	O	O	O	O	O
17. I bully other people (e.g. saying nasty things to them, hitting them)	O	O	O	O	O
18. I take my feelings out on objects around me (e.g. deliberately causing damage to my house or outdoor things)	O	O	O	O	O
19. Things feel unreal (e.g. I feel strange, things around me feel strange, I daydream)	O	O	O	O	O

PART B

This part asks how you **GENERALLY** respond to ***particular*** emotions.

1. **In GENERAL** when I feel the emotion SADNESS...

When I feel **SAD**...		Never	Seldom	Often	Very Often	Always
1.	**I try not to let myself feel or express this** (e.g. I pretend the situation doesn't exist, I keep the feeling locked up inside, I force myself to feel something else)	O	O	O	O	O
2.	**I seek social support** (e.g. I talk to someone about how I feel, I ask others for advice, I seek physical contact from others)	O	O	O	O	O
3.	**I change the way I view the situation** (e.g. I try to see a positive side, I put it into perspective, I review my goals or plans)	O	O	O	O	O
4.	**I take my feelings out on the people or objects around me** (e.g. I lash out, I bully people, I argue with people)	O	O	O	O	O

2. In **GENERAL** when I feel the emotion HAPPINESS...

When I feel **HAPPY**...		Never	Seldom	Often	Very Often	Always
1.	**I try not to let myself feel or express this** (e.g. I pretend the situation doesn't exist, I keep the feeling locked up inside, I force myself to feel something else)	O	O	O	O	O
2.	**I seek social support** (e.g. I talk to someone about how I feel, I ask others for advice, I seek physical contact from others)	O	O	O	O	O
3.	**I change the way I view the situation** (e.g. I try to see a positive side, I put it into perspective, I review my goals or plans)	O	O	O	O	O
4.	**I take my feelings out on the people or objects around me** (e.g. I lash out, I bully people, I argue with people)	O	O	O	O	O

3. **In GENERAL** when I feel the emotion SHAME...

When I feel **ASHAMED**...		Never	Seldom	Often	Very Often	Always
1.	**I try not to let myself feel or express this** (e.g. I pretend the situation doesn't exist, I keep the feeling locked up inside, I force myself to feel something else)	O	O	O	O	O
2.	**I seek social support** (e.g. I talk to someone about how I feel, I ask others for advice, I seek physical contact from others)	O	O	O	O	O
3.	**I change the way I view the situation** (e.g. I try to see a positive side, I put it into perspective, I review my goals or plans)	O	O	O	O	O
4.	**I take my feelings out on the people or objects around me** (e.g. I lash out, I bully people, I argue with people)	O	O	O	O	O

4. **In GENERAL** when I feel the emotion ANGER...

When I feel **ANGRY**...		Never	Seldom	Often	Very Often	Always
1.	**I try not to let myself feel or express this** (e.g. I pretend the situation doesn't exist, I keep the feeling locked up inside, I force myself to feel something else)	O	O	O	O	O
2.	**I seek social support** (e.g. I talk to someone about how I feel, I ask others for advice, I seek physical contact from others)	O	O	O	O	O
3.	**I change the way I view the situation** (e.g. I try to see a positive side, I put it into perspective, I review my goals or plans)	O	O	O	O	O
4.	**I take my feelings out on the people or objects around me** (e.g. I lash out, I bully people, I argue with people)	O	O	O	O	O

5. **In GENERAL** when I feel the emotion ANXIETY...

When I feel **ANXIOUS**...		Never	Seldom	Often	Very Often	Always
1.	**I try not to let myself feel or express this** (e.g. I pretend the situation doesn't exist, I keep the feeling locked up inside, I force myself to feel something else)	O	O	O	O	O
2.	**I seek social support** (e.g. I talk to someone about how I feel, I ask others for advice, I seek physical contact from others)	O	O	O	O	O
3.	**I change the way I view the situation** (e.g. I try to see a positive side, I put it into perspective, I review my goals or plans)	O	O	O	O	O
4.	**I take my feelings out on the people or objects around me** (e.g. I lash out, I bully people, I argue with people)	O	O	O	O	O

6. **In GENERAL** when I feel the emotion GUILT...

When I feel **GUILTY**...		Never	Seldom	Often	Very Often	Always
1.	**I try not to let myself feel or express this** (e.g. I pretend the situation doesn't exist, I keep the feeling locked up inside, I force myself to feel something else)	O	O	O	O	O
2.	**I seek social support** (e.g. I talk to someone about how I feel, I ask others for advice, I seek physical contact from others)	O	O	O	O	O
3.	**I change the way I view the situation** (e.g. I try to see a positive side, I put it into perspective, I review my goals or plans)	O	O	O	O	O
4.	**I take my feelings out on the people or objects around me** (e.g. I lash out, I bully people, I argue with people)	O	O	O	O	O

Self-Rating Questionnaire

STAXI Item Booklet

Instructions

In addition to this Item Booklet you should have a STAXI Rating Sheet. This booklet is divided into three Sections. Each Section contains a number of statements that people have used to describe their feelings and behaviour. Please note that each Section has *different* directions. Carefully read the directions for each Section before recording your responses on the Rating Sheet.

There are no right or wrong answers. In responding to each statement, give the answer that describes you best. DO NOT ERASE! If you need to change your answer, make an "X" through the incorrect response and then fill in the correct one.

Examples

1. ① ② ③ ④

2. ① ② ③ ④

Section 1 Directions

A number of statements that people use to describe themselves are given below. Read each statement and than fill in the circle with the number that indicates how you feel *right now*. Remember that there are no right or wrong answers. Do not spend too much time on any one statement, but give the answer which seems to *best* describe your *present feelings*.

Fill in ① for *Not at all*	Fill in ③ for *Moderately so*
Fill in ② for *Somewhat*	Fill in ④ for *Very much so*

How I Feel Right Now

1. I am furious.
2. I feel irritated.
3. I feel angry.
4. I feel like yelling at somebody.
5. I feel like breaking things.
6. I am mad.
7. I feel like banging on the table.
8. I feel like hitting someone.
9. I am burned up.
10. I feel like swearing.

Section 2 Directions

A number of statements that people use to describe themselves are given below. Read each statement and then fill in the circle with the number which indicates how you *generally feel*. Remember that there are no right or wrong answers. Do not spend too much time on any one statement, but give the answer which seems to *best* describe how you *generally feel*.

Fill in ① for *Almost never*	Fill in ③ for *Often*
Fill in ② for *Sometimes*	Fill in ④ for *Almost always*

How I Generally Feel

11. I am quick tempered.
12. I have a fiery temper.
13. I am a hot-headed person.
14. I get angry when I'm slowed down by others' mistakes.
15. I feel annoyed when I am not give recognition for doing good work.
16. I fly off the handle.
17. When I get mad I say nasty things.
18. It makes me furious when I am criticised in front of others.
19. When I get frustrated, I feel like hitting someone.
20. I feel infuriated when I do a good job and get a poor evaluation.

Section 3 Directions

Everyone feels angry or furious from time to time, but people differ in the ways that they react when they are angry. A number of statements are listed below which people use to describe their reactions when they feel *angry* or *furious*. Read each statement and then fill in the circle with the number that indicates how *often* you *generally* react or behave in the manner described when you feel angry or furious. Remember that there are no right or wrong answers. Do not spend too much time on any one statement.

Fill in ① for *Almost never*	Fill in ③ for *Often*
Fill in ② for *Sometimes*	Fill in ④ for *Almost always*

When Angry or Furious...

21. I control my temper.
22. I express my anger.
23. I keep things in.
24 I am patient with others.
25. I pout or sulk.
26. I withdraw from people.
27. I make sarcastic remarks to others.
28. I keep my cool.
29. I do things like slam doors.
30. I boil inside, but don't show it.
31. I control my behaviour.
32. I argue with others.
33. I tend to harbour grudges that I don't tell anyone about.
34. I strike out at whatever infuriates me.
35. I can stop myself from losing my temper.
36. I am secretly quite critical of others.
37. I am angrier than I am willing to admit.
38. I calm down faster than most people.
39. I say nasty things.
40. I try to be tolerant and understanding.
41. I'm irritated a great deal more than people are aware of.
42. I lose my temper.
43. If someone annoys me, I'm apt to tell him or her how I feel.
44. I control my angry feelings.

Self-Rating Questionnaire

STAXI Rating Sheet

PART 1

How I Feel Right Now

1.	①	②	③	④
2.	①	②	③	④
3.	①	②	③	④
4.	①	②	③	④
5.	①	②	③	④
6.	①	②	③	④
7.	①	②	③	④
8.	①	②	③	④
9.	①	②	③	④
10.	①	②	③	④

PART 2

How I Generally Feel

11.	①	②	③	④
12.	①	②	③	④
13.	①	②	③	④
14.	①	②	③	④
15.	①	②	③	④
16.	①	②	③	④
17.	①	②	③	④
18.	①	②	③	④
19.	①	②	③	④
20.	①	②	③	④

PART 3

When Angry or Furious

21.	①	②	③	④
22.	①	②	③	④
23.	①	②	③	④
24.	①	②	③	④
25.	①	②	③	④
26.	①	②	③	④
27.	①	②	③	④
28.	①	②	③	④
29.	①	②	③	④
30.	①	②	③	④
31.	①	②	③	④
32.	①	②	③	④
33.	①	②	③	④
34.	①	②	③	④
35.	①	②	③	④
36.	①	②	③	④
37.	①	②	③	④
38.	①	②	③	④
39.	①	②	③	④
40.	①	②	③	④
41.	①	②	③	④
42.	①	②	③	④
43.	①	②	③	④
44.	①	②	③	④

BDI-II

Date:

ame: _____ Marital Status: _____ Age: _____ Sex: _____

ccupation: _____ Education: _____

istructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and en pick out the **one statement** in each group that best describes the way you have been feeling during the **past two eeks, including today**. Circle the number beside the statement you have picked. If several statements in the group eem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one atement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness

0 I do not feel sad.
1 I feel sad much of the time.
2 I am sad all the time.
3 I am so sad or unhappy that I can't stand it.

2. Pessimism

0 I am not discouraged about my future.
1 I feel more discouraged about my future than I used to be.
2 I do not expect things to work out for me.
3 I feel my future is hopeless and will only get worse.

3. Past Failure

0 I do not feel like a failure.
1 I have failed more than I should have.
2 As I look back, I see a lot of failures.
3 I feel I am a total failure as a person.

4. Loss of Pleasure

0 I get as much pleasure as I ever did from the things I enjoy.
1 I don't enjoy things as much as I used to.
2 I get very little pleasure from the things I used to enjoy.
3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

0 I don't feel particularly guilty.
1 I feel guilty over many things I have done or should have done.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6. Punishment Feelings

0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7. Self-Dislike

0 I feel the same about myself as ever.
1 I have lost confidence in myself.
2 I am disappointed in myself.
3 I dislike myself.

8. Self-Criticalness

0 I don't criticize or blame myself more than usual.
1 I am more critical of myself than I used to be.
2 I criticize myself for all of my faults.
3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. Crying

0 I don't cry anymore than I used to.
1 I cry more than I used to.
2 I cry over every little thing.
3 I feel like crying, but I can't.

Subtotal Page 1

Continued on Back

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11. Agitation

0 I am no more restless or wound up than usual.

1 I feel more restless or wound up than usual.

2 I am so restless or agitated that it's hard to stay still.

3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.

1 I am less interested in other people or things than before.

2 I have lost most of my interest in other people or things.

3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.

1 I find it more difficult to make decisions than usual.

2 I have much greater difficulty in making decisions than I used to.

3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.

1 I don't consider myself as worthwhile and useful as I used to.

2 I feel more worthless as compared to other people.

3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.

1 I have less energy than I used to have.

2 I don't have enough energy to do very much.

3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.

1a I sleep somewhat more than usual.

1b I sleep somewhat less than usual.

2a I sleep a lot more than usual.

2b I sleep a lot less than usual.

3a I sleep most of the day.

3b I wake up 1–2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable than usual.

1 I am more irritable than usual.

2 I am much more irritable than usual.

3 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.

1a My appetite is somewhat less than usual.

1b My appetite is somewhat greater than usual.

2a My appetite is much less than before.

2b My appetite is much greater than usual.

3a I have no appetite at all.

3b I crave food all the time.

19. Concentration Difficulty

0 I can concentrate as well as ever.

1 I can't concentrate as well as usual.

2 It's hard to keep my mind on anything for very long.

3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.

1 I get more tired or fatigued more easily than usual.

2 I am too tired or fatigued to do a lot of the things I used to do.

3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.

1 I am less interested in sex than I used to be.

2 I am much less interested in sex now.

3 I have lost interest in sex completely.

10 11 12 A B C D E

_____ Subtotal Page 2

_____ Subtotal Page 1

_____ Total Score

®

ME _____ DATE _____

ow is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by each ptom during the PAST WEEK, INCLUDING TODAY, by placing an X in the corresponding space in the column next to each symptom.

	NOT AT ALL	MILDLY but it didn't bother me much	MODERATELY It was very unpleasant but I could stand it	SEVERELY I could barely stand it
1. Numbness or tingling.				
2. Feeling hot.				
3. Wobbliness in legs.				
4. Unable to relax.				
5. Fear of the worst happening.				
6. Dizzy or lightheaded.				
7. Heart pounding or racing.				
8. Unsteady.				
9. Terrified.				
10. Nervous.				
11. Feelings of choking.				
12. Hands trembling.				
13. Shaky.				
14. Fear of losing control.				
15. Difficulty breathing.				
16. Fear of dying.				
17. Scared.				
18. Indigestion or discomfort in abdomen.				
19. Faint.				
20. Face flushed.				
21. Sweating (not due to heat).				

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Appendix IV

MVA

Univariate Statistics

	N	Mean	Std. Deviation	Missing		No. of Extremes[a,b]	
				Count	Percent	Low	High
AGE	104	41.2692	13.6433	0	.0	0	0
DCS1	100	8.6711	12.8053	4	3.8	0	7
DCS2MIG	104	.1923	.3960	0	.0	.	.
DCS2TT	104	.0962	.2962	0	.0	.	.
DCS2COMB	104	.1923	.3960	0	.0	.	.
DCS2CLUS	104	.0769	.2678	0	.0	.	.
DCS2OTHR	104	.0385	.1932	0	.0	.	.
DCSVAS1	104	2.1923	3.1564	0	.0	0	5
DCSVAS2	104	3.7788	3.6945	0	.0	0	0
HDIFREQ	101	1.6238	1.2795	3	2.9	0	0
HDISEVTY	98	1.6224	1.1886	6	5.8	0	0
ERQSAD1	104	1.3173	.9681	0	.0	0	3
ERQSAD2	104	1.7115	1.0943	0	.0	0	7
ERQSAD3	104	1.9808	.9241	0	.0	0	0
ERQSAD4	104	.4808	.7880	0	.0	0	2
ERQHAP1	104	.3750	.8266	0	.0	.	.
ERQHAP2	104	1.5288	1.1984	0	.0	0	0
ERQHAP3	104	1.4327	1.1555	0	.0	0	0
ERQHAP4	103	.2233	.6093	1	1.0	.	.
ERQASH1	103	1.5049	1.1191	1	1.0	0	6
ERQASH2	103	1.2621	.9697	1	1.0	0	3
ERQASH3	103	1.8252	.8680	1	1.0	0	3
ERQASH4	103	.3689	.6566	1	1.0	0	1
ERQANG1	103	1.5825	.9448	1	1.0	0	2
ERQANG2	103	1.5825	1.0340	1	1.0	0	5
ERQANG3	103	1.9029	.7985	1	1.0	0	3
ERQANG4	103	.6602	.8581	1	1.0	0	4
ERQANX1	103	1.4951	.9587	1	1.0	0	3
ERQANX2	102	1.7059	1.0492	2	1.9	0	6
ERQANX3	103	1.8932	.8274	1	1.0	0	2
ERQANX4	103	.5049	.8503	1	1.0	0	3
ERQGLT1	103	1.2136	.9461	1	1.0	0	1
ERQGLT2	102	1.4510	1.0866	2	1.9	0	5
ERQGLT3	103	1.7767	.9593	1	1.0	0	4
ERQGLT4	103	.2718	.5276	1	1.0	.	.
BDITOT	101	11.04	8.70	3	2.9	0	2
BAITOT	102	8.84	7.83	2	1.9	0	6
HDITOT	104	28.35	26.94	0	.0	0	0
HDIEMO	104	11.62	12.62	0	.0	0	0
HDIFUN	104	16.73	15.25	0	.0	0	0
EXFUNTOT	104	7.49	2.84	0	.0	0	0
INFUNTOT	104	10.13	2.76	0	.0	1	2
EXDYSTOT	104	17.50	1.99	0	.0	0	0
INDYSTOT	104	14.29	3.03	0	.0	2	0
STANGER	100	11.43	2.92	4	3.8	0	18
TANGER	100	17.71	4.33	4	3.8	0	1
TANGERT	100	6.33	2.38	4	3.8	0	1
TANGERR	100	8.51	2.12	4	3.8	0	0
ANGERIN	100	16.76	4.23	4	3.8	0	0
ANGOUT	100	14.17	3.17	4	3.8	0	3
ANGCON	100	21.90	5.28	4	3.8	0	0
ANGEXP	100	25.0300	8.0722	4	3.8	0	0
BESANGPW	104	11.8942	4.9833	0	.0	0	0

Univariate Statistics

	N	Mean	Std. Deviation	Missing		No. of Extremes[a,b]	
				Count	Percent	Low	High
BESSADPW	104	8.2115	4.4453	0	.0	0	3
BESDISPW	104	6.5096	3.4976	0	.0	0	5
BESFRPW	104	13.7981	5.2424	0	.0	0	0
BESHAPPW	104	17.6250	5.1656	0	.0	0	0
BESANGGN	104	12.7404	4.5730	0	.0	0	1
BESSADGN	104	8.5385	4.3710	0	.0	0	4
BESDISGN	104	7.3365	4.1818	0	.0	0	5
BESFRGN	104	14.6154	4.7117	0	.0	0	0
BESHAPGN	104	19.2500	5.4215	0	.0	2	0
BESANGCP	93	13.0108	4.7947	11	10.6	0	1
BESSADCP	93	11.5914	5.1420	11	10.6	0	0
BESDISCP	93	12.8280	6.0390	11	10.6	0	0
BESFRCP	93	14.6129	5.3202	11	10.6	0	0
BESHAPCP	93	6.5161	3.3154	11	10.6	0	4
SEX	104			0	.0		
EDUC	68			36	34.6		
OCCUP	97			7	6.7		
MTSTATUS	97			7	6.7		

a. Number of cases outside the range (Q1 - 1.5*IQR, Q3 + 1.5*IQR).

b. . indicates that the inter quartile range (IQR) is zero.

Statistics

		BDI-II	BAI	HDI total	HDI Emotional	HDI Functional	BES Anger (past week)	BES Sadness (past week)	BES Disgust (past week)
N	Valid	101	102	104	104	104	104	104	104
	Missing	3	2	0	0	0	0	0	0
Mean		11.04	8.84	28.35	11.62	16.73	11.8942	8.2115	6.5096
Std. Deviation		8.703	7.826	26.939	12.616	15.250	4.98331	4.44529	3.49756
Skewness		1.150	1.462	.447	.790	.309	.558	1.261	1.947
Std. Error of Skewness		.240	.239	.237	.237	.237	.237	.237	.237
Kurtosis		1.514	1.946	-1.219	-.654	-1.359	-.526	.914	4.071
Std. Error of Kurtosis		.476	.474	.469	.469	.469	.469	.469	.469

Statistics

		BES Fear (past week)	BES Happiness (past week)	BES Anger (in general)	BES Sadness (in general)	BES Disgust (in general)	BES Fear (in general)	BES Happiness (in general)
N	Valid	104	104	104	104	104	104	104
	Missing	0	0	0	0	0	0	0
Mean		13.7981	17.6250	12.7404	8.5385	7.3365	14.6154	19.2500
Std. Deviation		5.24243	5.16558	4.57302	4.37096	4.18182	4.71170	5.42155
Skewness		.156	-.666	.755	1.501	2.083	.268	-.853
Std. Error of Skewness		.237	.237	.237	.237	.237	.237	.237
Kurtosis		-.826	-.099	.208	2.414	5.206	-.571	.582
Std. Error of Kurtosis		.469	.469	.469	.469	.469	.469	.469

Statistics

		BES Anger (coping)	BES Sadness (coping)	BES Disgust (coping)	BES Fear (coping)	BES Happiness (coping)	ERQ-EF	ERQ-IF	ERQ-ED
N	Valid	93	93	93	93	93	104	104	104
	Missing	11	11	11	11	11	0	0	0
Mean		13.0108	11.5914	12.8280	14.6129	6.5161	7.49	10.13	17.50
Std. Deviation		4.79469	5.14198	6.03905	5.32025	3.31536	2.839	2.756	1.985
Skewness		.254	.142	.304	.075	1.441	.093	.291	-.812
Std. Error of Skewness		.250	.250	.250	.250	.250	.237	.237	.237
Kurtosis		-.366	-.790	-.405	-.246	2.015	-.460	.097	.088
Std. Error of Kurtosis		.495	.495	.495	.495	.495	.469	.469	.469

Statistics

		ERQ-ID	sad- ID	sad-EF	sad-IF	sad-ED	happy-ID	happy-EF	happy-IF	happy-ED
N	Valid	104	104	104	104	104	104	104	104	103
	Missing	0	0	0	0	0	0	0	0	1
Mean		14.29	1.3173	1.7115	1.9808	.4808	.3750	1.5288	1.4327	.2233
Std. Deviation		3.026	.96808	1.09432	.92412	.78803	.82659	1.19835	1.15552	.60928
Skewness		-.852	.631	.327	.114	1.824	2.670	.328	.167	3.558
Std. Error of Skewness		.237	.237	.237	.237	.237	.237	.237	.237	.238
Kurtosis		1.176	.265	-.509	.022	3.644	7.471	-.733	-1.001	15.587
Std. Error of Kurtosis		.469	.469	.469	.469	.469	.469	.469	.469	.472

Statistics

		ashamed-ID	ashamed-EF	ashamed-IF	ashamed-ED	angry-ID	angry-EF	angry-IF	angry-ED
N	Valid	103	103	103	103	103	103	103	103
	Missing	1	1	1	1	1	1	1	1
Mean		1.5049	1.2621	1.8252	.3689	1.5825	1.5825	1.9029	.6602
Std. Deviation		1.11912	.96975	.86803	.65664	.94479	1.03397	.79846	.85810
Skewness		.480	.698	.166	1.771	.291	.426	.295	1.386
Std. Error of Skewness		.238	.238	.238	.238	.238	.238	.238	.238
Kurtosis		-.411	.392	.012	2.624	-.381	-.212	.188	1.909
Std. Error of Kurtosis		.472	.472	.472	.472	.472	.472	.472	.472

Statistics

		anxious-ID	anxious-EF	anxious-IF	anxious-ED	guilty-ID	guilty-EF	guilty-IF	guilty-ED
N	Valid	103	102	103	103	103	102	103	103
	Missing	1	2	1	1	1	2	1	1
Mean		1.4951	1.7059	1.8932	.5049	1.2136	1.4510	1.7767	.2718
Std. Deviation		.95869	1.04917	.82738	.85030	.94610	1.08662	.95929	.52756
Skewness		.354	.356	.097	2.326	.620	.435	-.079	1.823
Std. Error of Skewness		.238	.239	.238	.238	.238	.239	.238	.238
Kurtosis		-.025	-.373	-.238	6.530	-.106	-.299	.058	2.512
Std. Error of Kurtosis		.472	.474	.472	.472	.472	.474	.472	.472

Statistics

		State Anger	Trait Anger	Trait Anger (temperament)	Trait Anger (reactive)	Anger-in	Anger-out	Anger-Control	Anger Expression
N	Valid	100	100	100	100	100	100	100	100
	Missing	4	4	4	4	4	4	4	4
Mean		11.43	17.71	6.33	8.51	16.76	14.17	21.90	25.0300
Std. Deviation		2.917	4.331	2.383	2.120	4.231	3.166	5.283	8.07222
Skewness		2.710	.480	1.075	-.235	.373	.621	-.170	.266
Std. Error of Skewness		.241	.241	.241	.241	.241	.241	.241	.241
Kurtosis		7.233	.418	.617	.124	-.102	.837	-.808	-.420
Std. Error of Kurtosis		.478	.478	.478	.478	.478	.478	.478	.478

Correlations – Headache Group

Correlations

		hdi-frequency head	level pain average	HDITOT	BDITOT
hdi-frequency head	Pearson Correlation	1	-.019	.087	.133
	Sig. (2-tailed)	.	.893	.531	.342
	N	54	54	54	53
level pain average	Pearson Correlation	-.019	1	.228	.124
	Sig. (2-tailed)	.893	.	.089	.368
	N	54	57	57	55
HDITOT	Pearson Correlation	.087	.228	1	.663**
	Sig. (2-tailed)	.531	.089	.	.000
	N	54	57	57	55
BDITOT	Pearson Correlation	.133	.124	.663**	1
	Sig. (2-tailed)	.342	.368	.000	.
	N	53	55	55	55
BAITOT	Pearson Correlation	.097	.185	.490**	.608**
	Sig. (2-tailed)	.484	.173	.000	.000
	N	54	56	56	55
EXFUNTOT	Pearson Correlation	.028	.099	-.147	-.149
	Sig. (2-tailed)	.841	.465	.274	.276
	N	54	57	57	55
INFUNTOT	Pearson Correlation	.087	.018	-.052	-.184
	Sig. (2-tailed)	.530	.897	.699	.179
	N	54	57	57	55
EXDYSTOT	Pearson Correlation	-.226	.161	.362**	.294*
	Sig. (2-tailed)	.100	.232	.006	.029
	N	54	57	57	55
INDYSTOT	Pearson Correlation	-.051	.349**	.592**	.469**
	Sig. (2-tailed)	.715	.008	.000	.000
	N	54	57	57	55
STANGER	Pearson Correlation	-.259	.040	.356**	.388**
	Sig. (2-tailed)	.058	.769	.007	.003
	N	54	56	56	55
TANGER	Pearson Correlation	-.075	-.116	.510**	.482**
	Sig. (2-tailed)	.588	.395	.000	.000
	N	54	56	56	55
TANGERT	Pearson Correlation	.008	.004	.427**	.466**
	Sig. (2-tailed)	.952	.975	.001	.000
	N	54	56	56	55
TANGERR	Pearson Correlation	-.170	-.228	.337*	.255
	Sig. (2-tailed)	.220	.091	.011	.060
	N	54	56	56	55
ANGERIN	Pearson Correlation	-.106	.055	.271*	.336*
	Sig. (2-tailed)	.445	.690	.043	.012
	N	54	56	56	55
ANGOUT	Pearson Correlation	-.007	-.064	.340*	.189
	Sig. (2-tailed)	.960	.639	.010	.168
	N	54	56	56	55
ANGCON	Pearson Correlation	.006	-.071	-.395**	-.301*
	Sig. (2-tailed)	.967	.603	.003	.025
	N	54	56	56	55
ANGEXP	Pearson Correlation	-.058	.047	.496**	.413**
	Sig. (2-tailed)	.678	.731	.000	.002
	N	54	56	56	55
BESANGPW	Pearson Correlation	-.216	.103	.596**	.599**
	Sig. (2-tailed)	.116	.445	.000	.000
	N	54	57	57	55

Correlations

		hdi-frequency head	level pain average	HDITOT	BDITOT
BESSADPW	Pearson Correlation	-.136	.261*	.581**	.641**
	Sig. (2-tailed)	.327	.050	.000	.000
	N	54	57	57	55
BESDISPW	Pearson Correlation	-.155	.166	.422**	.562**
	Sig. (2-tailed)	.263	.216	.001	.000
	N	54	57	57	55
BESFRPW	Pearson Correlation	-.050	.094	.476**	.567**
	Sig. (2-tailed)	.719	.488	.000	.000
	N	54	57	57	55
BESHAPPW	Pearson Correlation	.282*	.022	-.333*	-.473**
	Sig. (2-tailed)	.039	.872	.011	.000
	N	54	57	57	55
BESANGGN	Pearson Correlation	-.151	.197	.632**	.528**
	Sig. (2-tailed)	.275	.142	.000	.000
	N	54	57	57	55
BESSADGN	Pearson Correlation	-.055	.299*	.648**	.631**
	Sig. (2-tailed)	.691	.024	.000	.000
	N	54	57	57	55
BESDISGN	Pearson Correlation	-.062	.067	.379**	.420**
	Sig. (2-tailed)	.657	.618	.004	.001
	N	54	57	57	55
BESFRGN	Pearson Correlation	.042	.196	.463**	.508**
	Sig. (2-tailed)	.763	.144	.000	.000
	N	54	57	57	55
BESHAPGN	Pearson Correlation	.228	.089	-.307*	-.532**
	Sig. (2-tailed)	.097	.512	.020	.000
	N	54	57	57	55
BESANGCP	Pearson Correlation	-.155	.134	.528**	.533**
	Sig. (2-tailed)	.315	.369	.000	.000
	N	44	47	47	45
BESSADCP	Pearson Correlation	.120	.184	.528**	.560**
	Sig. (2-tailed)	.438	.215	.000	.000
	N	44	47	47	45
BESDISCP	Pearson Correlation	.117	.013	.267	.317*
	Sig. (2-tailed)	.450	.930	.069	.034
	N	44	47	47	45
BESFRCP	Pearson Correlation	.245	.061	.440**	.394**
	Sig. (2-tailed)	.110	.685	.002	.007
	N	44	47	47	45
BESHAPCP	Pearson Correlation	.067	.116	.376**	.356*
	Sig. (2-tailed)	.664	.437	.009	.016
	N	44	47	47	45
anger-ID	Pearson Correlation	-.109	.017	.301*	.181
	Sig. (2-tailed)	.432	.901	.023	.187
	N	54	57	57	55
anger-EF	Pearson Correlation	.105	-.023	-.146	-.037
	Sig. (2-tailed)	.448	.867	.280	.789
	N	54	57	57	55
anger-IF	Pearson Correlation	.013	-.199	-.358**	-.282*
	Sig. (2-tailed)	.926	.138	.006	.037
	N	54	57	57	55
anger-ED	Pearson Correlation	-.087	.029	.325*	.219
	Sig. (2-tailed)	.534	.830	.014	.108
	N	54	57	57	55

Correlations

		hdi-frequency head	level pain average	HDITOT	BDITOT
sad- ID	Pearson Correlation	-.104	.164	.398**	.269*
	Sig. (2-tailed)	.455	.222	.002	.047
	N	54	57	57	55
sad-EF	Pearson Correlation	.104	-.097	-.222	-.151
	Sig. (2-tailed)	.454	.474	.098	.272
	N	54	57	57	55
sad-IF	Pearson Correlation	-.035	-.053	-.188	-.308*
	Sig. (2-tailed)	.802	.694	.162	.022
	N	54	57	57	55
sad-ED	Pearson Correlation	-.047	.083	.339**	.175
	Sig. (2-tailed)	.737	.540	.010	.202
	N	54	57	57	55
happy-ID	Pearson Correlation	-.098	.167	.291*	.222
	Sig. (2-tailed)	.482	.214	.028	.103
	N	54	57	57	55
happy-EF	Pearson Correlation	-.090	-.034	-.155	-.234
	Sig. (2-tailed)	.516	.802	.248	.086
	N	54	57	57	55
happy-IF	Pearson Correlation	-.080	.051	-.066	-.079
	Sig. (2-tailed)	.565	.706	.627	.565
	N	54	57	57	55
happy-ED	Pearson Correlation	-.204	.048	.134	.185
	Sig. (2-tailed)	.138	.723	.321	.175
	N	54	57	57	55
ashamed-ID	Pearson Correlation	-.173	-.033	.116	-.039
	Sig. (2-tailed)	.211	.808	.391	.778
	N	54	57	57	55
ashamed-EF	Pearson Correlation	.092	-.053	-.122	-.093
	Sig. (2-tailed)	.509	.696	.366	.501
	N	54	57	57	55
ashamed-IF	Pearson Correlation	-.024	-.193	-.225	-.192
	Sig. (2-tailed)	.863	.150	.092	.160
	N	54	57	57	55
ashamed-ED	Pearson Correlation	-.189	.030	.278*	.188
	Sig. (2-tailed)	.171	.825	.036	.169
	N	54	57	57	55
anxious-ID	Pearson Correlation	-.206	.190	.023	-.096
	Sig. (2-tailed)	.136	.156	.867	.487
	N	54	57	57	55
anxious-EF	Pearson Correlation	.091	-.072	-.209	-.061
	Sig. (2-tailed)	.518	.596	.122	.659
	N	53	56	56	54
anxious-IF	Pearson Correlation	-.143	-.076	-.269*	-.224
	Sig. (2-tailed)	.304	.577	.043	.100
	N	54	57	57	55
anxious-ED	Pearson Correlation	.011	.008	.374**	.233
	Sig. (2-tailed)	.939	.955	.004	.087
	N	54	57	57	55
guilty-ID	Pearson Correlation	.022	-.031	.083	.030
	Sig. (2-tailed)	.877	.819	.542	.826
	N	54	57	57	55
guilty-EF	Pearson Correlation	.146	-.063	-.146	-.174
	Sig. (2-tailed)	.296	.645	.284	.207
	N	53	56	56	54

Correlations

		hdi-frequency head	level pain average	HDITOT	BDITOT
guilty-IF	Pearson Correlation	-.074	-.085	-.194	-.239
	Sig. (2-tailed)	.596	.531	.149	.079
	N	54	57	57	55
guilty-ED	Pearson Correlation	-.040	-.057	.208	.159
	Sig. (2-tailed)	.773	.671	.120	.245
	N	54	57	57	55

Correlations – Control Group

Correlations

		hdi-frequency head	level pain average	HDITOT	BDITOT
hdi-frequency head	Pearson Correlation	1	.352*	.445**	.190
	Sig. (2-tailed)	.	.015	.002	.205
	N	47	47	47	46
level pain average	Pearson Correlation	.352*	1	.541**	.242
	Sig. (2-tailed)	.015	.	.000	.105
	N	47	47	47	46
HDITOT	Pearson Correlation	.445**	.541**	1	.424**
	Sig. (2-tailed)	.002	.000	.	.003
	N	47	47	47	46
BDITOT	Pearson Correlation	.190	.242	.424**	1
	Sig. (2-tailed)	.205	.105	.003	.
	N	46	46	46	46
BAITOT	Pearson Correlation	.181	.361*	.346*	.591**
	Sig. (2-tailed)	.230	.014	.018	.000
	N	46	46	46	46
EXFUNTOT	Pearson Correlation	.068	.067	-.129	.058
	Sig. (2-tailed)	.651	.653	.389	.700
	N	47	47	47	46
INFUNTOT	Pearson Correlation	.103	-.186	-.156	.077
	Sig. (2-tailed)	.492	.211	.296	.610
	N	47	47	47	46
EXDYSTOT	Pearson Correlation	.411**	.245	.481**	.240
	Sig. (2-tailed)	.004	.097	.001	.108
	N	47	47	47	46
INDYSTOT	Pearson Correlation	.224	.217	.447**	.619**
	Sig. (2-tailed)	.129	.143	.002	.000
	N	47	47	47	46
STANGER	Pearson Correlation	.168	.088	-.014	.137
	Sig. (2-tailed)	.277	.571	.928	.376
	N	44	44	44	44
TANGER	Pearson Correlation	.114	.131	.227	.242
	Sig. (2-tailed)	.463	.396	.138	.114
	N	44	44	44	44
TANGERT	Pearson Correlation	.121	.007	.169	.080
	Sig. (2-tailed)	.433	.965	.273	.604
	N	44	44	44	44
TANGERR	Pearson Correlation	.036	.119	.158	.201
	Sig. (2-tailed)	.817	.441	.306	.191
	N	44	44	44	44
ANGERIN	Pearson Correlation	.007	.122	.148	.418**
	Sig. (2-tailed)	.965	.431	.339	.005
	N	44	44	44	44
ANGOUT	Pearson Correlation	.228	.420**	.552**	.214
	Sig. (2-tailed)	.137	.004	.000	.164
	N	44	44	44	44
ANGCON	Pearson Correlation	-.048	.067	-.327*	-.045
	Sig. (2-tailed)	.758	.664	.030	.772
	N	44	44	44	44
ANGEXP	Pearson Correlation	.143	.220	.584**	.392**
	Sig. (2-tailed)	.353	.152	.000	.008
	N	44	44	44	44
BESANGPW	Pearson Correlation	.218	.059	.087	.144
	Sig. (2-tailed)	.140	.694	.563	.341
	N	47	47	47	46

Correlations

		hdi-frequency head	level pain average	HDITOT	BDITOT
BESSADPW	Pearson Correlation	-.106	.076	.157	.455**
	Sig. (2-tailed)	.478	.610	.291	.001
	N	47	47	47	46
BESDISPW	Pearson Correlation	-.068	.092	.156	.334*
	Sig. (2-tailed)	.648	.540	.297	.023
	N	47	47	47	46
BESFRPW	Pearson Correlation	.045	.195	-.090	.415**
	Sig. (2-tailed)	.762	.190	.549	.004
	N	47	47	47	46
BESHAPPW	Pearson Correlation	-.050	-.129	-.002	-.048
	Sig. (2-tailed)	.738	.388	.987	.750
	N	47	47	47	46
BESANGGN	Pearson Correlation	.283	.177	.311*	.476**
	Sig. (2-tailed)	.054	.233	.033	.001
	N	47	47	47	46
BESSADGN	Pearson Correlation	.120	.154	.191	.511**
	Sig. (2-tailed)	.423	.302	.198	.000
	N	47	47	47	46
BESDISGN	Pearson Correlation	.045	.235	.244	.457**
	Sig. (2-tailed)	.763	.112	.099	.001
	N	47	47	47	46
BESFRGN	Pearson Correlation	.254	.276	.107	.351*
	Sig. (2-tailed)	.085	.060	.473	.017
	N	47	47	47	46
BESHAPGN	Pearson Correlation	-.046	-.109	-.014	.109
	Sig. (2-tailed)	.759	.465	.924	.471
	N	47	47	47	46
BESANGCP	Pearson Correlation	.166	-.032	.013	.208
	Sig. (2-tailed)	.270	.833	.934	.170
	N	46	46	46	45
BESSADCP	Pearson Correlation	.164	.150	.151	.185
	Sig. (2-tailed)	.275	.319	.317	.225
	N	46	46	46	45
BESDISCP	Pearson Correlation	.141	.039	.101	.215
	Sig. (2-tailed)	.349	.797	.502	.156
	N	46	46	46	45
BESFRCP	Pearson Correlation	.281	.321*	.175	.427**
	Sig. (2-tailed)	.059	.030	.246	.003
	N	46	46	46	45
BESHAPCP	Pearson Correlation	-.087	.096	.073	.243
	Sig. (2-tailed)	.563	.528	.629	.108
	N	46	46	46	45
anger-ID	Pearson Correlation	-.133	-.042	-.170	.037
	Sig. (2-tailed)	.379	.781	.258	.809
	N	46	46	46	45
anger-EF	Pearson Correlation	.175	-.180	-.201	.038
	Sig. (2-tailed)	.246	.231	.181	.803
	N	46	46	46	45
anger-IF	Pearson Correlation	.045	-.259	-.163	.068
	Sig. (2-tailed)	.767	.082	.279	.656
	N	46	46	46	45
anger-ED	Pearson Correlation	.263	.241	.364*	.203
	Sig. (2-tailed)	.077	.107	.013	.181
	N	46	46	46	45

Correlations

		hdi-frequency head	level pain average	HDITOT	BDITOT
sad- ID	Pearson Correlation	.006	-.099	.172	.358*
	Sig. (2-tailed)	.965	.506	.248	.014
	N	47	47	47	46
sad-EF	Pearson Correlation	.163	-.001	-.149	-.072
	Sig. (2-tailed)	.272	.993	.318	.636
	N	47	47	47	46
sad-IF	Pearson Correlation	.197	-.213	-.115	-.150
	Sig. (2-tailed)	.184	.151	.443	.321
	N	47	47	47	46
sad-ED	Pearson Correlation	.402**	-.023	.311*	.203
	Sig. (2-tailed)	.005	.876	.034	.175
	N	47	47	47	46
happy-ID	Pearson Correlation	-.016	.063	.173	.021
	Sig. (2-tailed)	.917	.673	.246	.891
	N	47	47	47	46
happy-EF	Pearson Correlation	.289*	.188	-.134	.161
	Sig. (2-tailed)	.049	.206	.369	.284
	N	47	47	47	46
happy-IF	Pearson Correlation	.086	.045	.014	.189
	Sig. (2-tailed)	.567	.764	.925	.207
	N	47	47	47	46
happy-ED	Pearson Correlation	.136	-.095	.030	.180
	Sig. (2-tailed)	.369	.528	.841	.238
	N	46	46	46	45
ashamed-ID	Pearson Correlation	-.008	.090	.015	.047
	Sig. (2-tailed)	.960	.551	.921	.760
	N	46	46	46	45
ashamed-EF	Pearson Correlation	-.009	.050	-.104	.048
	Sig. (2-tailed)	.953	.742	.490	.754
	N	46	46	46	45
ashamed-IF	Pearson Correlation	.187	-.169	-.152	-.057
	Sig. (2-tailed)	.213	.260	.313	.711
	N	46	46	46	45
ashamed-ED	Pearson Correlation	.157	-.151	.152	.160
	Sig. (2-tailed)	.296	.318	.314	.293
	N	46	46	46	45
anxious-ID	Pearson Correlation	-.077	-.147	-.144	.200
	Sig. (2-tailed)	.612	.331	.341	.187
	N	46	46	46	45
anxious-EF	Pearson Correlation	.106	-.047	-.082	-.135
	Sig. (2-tailed)	.483	.758	.590	.377
	N	46	46	46	45
anxious-IF	Pearson Correlation	.042	-.121	-.242	.026
	Sig. (2-tailed)	.783	.424	.106	.867
	N	46	46	46	45
anxious-ED	Pearson Correlation	.420**	.154	.509**	.293
	Sig. (2-tailed)	.004	.308	.000	.051
	N	46	46	46	45
guilty-ID	Pearson Correlation	.130	.032	.098	.269
	Sig. (2-tailed)	.390	.835	.518	.074
	N	46	46	46	45
guilty-EF	Pearson Correlation	.165	.033	-.047	.143
	Sig. (2-tailed)	.274	.828	.756	.350
	N	46	46	46	45

Correlations

		hdi-frequency head	level pain average	HDITOT	BDITOT
guilty-IF	Pearson Correlation	.157	-.046	-.056	.042
	Sig. (2-tailed)	.298	.762	.711	.784
	N	46	46	46	45
guilty-ED	Pearson Correlation	.194	-.128	.140	.093
	Sig. (2-tailed)	.197	.396	.354	.543
	N	46	46	46	45